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DB Name	Query	Hit Count	Set Name
USPT,JPAB,EPAB,DWPI	trell and (peptide or polypeptide or protein)	4	L8
USPT,JPAB,EPAB,DWPI	trell and (tumor or tumour)	2	L7
USPT,JPAB,EPAB,DWPI	trell	83	L6
USPT,JPAB,EPAB,DWPI	l1 and trell	1	L5
USPT,JPAB,EPAB,DWPI	chichportiche-y$.in.	0	L4
USPT,JPAB,EPAB,DWPI	chicheportiche-y$.in.	1	L3
USPT,JPAB,EPAB,DWPI	chercheportiche-y$.in.	0	L2
USPT,JPAB,EPAB,DWPI	browning-j$.in.	329	L1

DB Name	Query	Hit Count	Set Name
USPT,JPAB,EPAB,DWPI	l17 and trell	0	L18
USPT,JPAB,EPAB,DWPI	l16 and related	779	L17
USPT,JPAB,EPAB,DWPI	l15 and family	847	L16
USPT,JPAB,EPAB,DWPI	l14 and (necrosis adj1 factor)	1658	L15
USPT,JPAB,EPAB,DWPI	l9 or l10 or l11 or l12 or l13	16573	L14
USPT,JPAB,EPAB,DWPI	((536/23.1 |536/23.5 |536/25.1)!.CCLS.)	6189	L13
USPT,JPAB,EPAB,DWPI	((424/93.2 |424/93.21 |424/93.7)!.CCLS.)	868	L12
USPT,JPAB,EPAB,DWPI	((530/350 |530/351)!.CCLS.)	5710	L11
USPT,JPAB,EPAB,DWPI	((514/44 |514/885)!.CCLS.)	1817	L10
USPT,JPAB,EPAB,DWPI	((435/69.1 |435/70.1 |435/70.3 |435/455 |435/325)!.CCLS.)	7869	L9
USPT,JPAB,EPAB,DWPI	trell and (peptide or polypeptide or protein)	4	L8
USPT,JPAB,EPAB,DWPI	trell and (tumor or tumour)	2	L7
USPT,JPAB,EPAB,DWPI	trell	83	L6
USPT,JPAB,EPAB,DWPI	l1 and trell	1	L5
USPT,JPAB,EPAB,DWPI	chichportiche-y$.in.	0	L4
USPT,JPAB,EPAB,DWPI	chicheportiche-y$.in.	1	L3
USPT,JPAB,EPAB,DWPI	chercheportiche-y$.in.	0	L2
USPT,JPAB,EPAB,DWPI	browning-j$.in.	329	L1

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on STN.

=> s review/dt

L1    1390059 REVIEW/DT

=> s l1 and (tumor or tumour)(w)(necrosis factor)/ab,bi

      201981 TUMOR
        449 TUMOUR
      44818 NECROSIS/AB
     475068 FACTOR/AB
      26300 (NECROSIS FACTOR)/AB
            ((NECROSIS(W)FACTOR)/AB)
      55889 NECROSIS/BI
     569092 FACTOR/BI
      34652 (NECROSIS FACTOR)/BI
            ((NECROSIS(W)FACTOR)/BI)
      34620 (TUMOR OR TUMOUR)(W)(NECROSIS
FACTOR)/AB,BI
L2      2613 L1 AND (TUMOR OR TUMOUR)(W)(NECROSIS
FACTOR)/AB,BI

=> s l2 and related/ab,bi

     574704 RELATED/AB
     656648 RELATED/BI
L3      206 L2 AND RELATED/AB,BI

=> s l3 and family/ab,bi

      97856 FAMILY/AB
     107175 FAMILY/BI
L4       42 L3 AND FAMILY/AB,BI

=> s l4 and function#/ab,bi

     900638 FUNCTION#/AB
    1053643 FUNCTION#/BI
L5       16 L4 AND FUNCTION#/AB,BI

=> s l5 and homology/ab,bi

        683 HOMOLOGY/AB
       5594 HOMOLOGY/BI
L6        0 L5 AND HOMOLOGY/AB,BI

=> d l5 1- bib ab

YOU HAVE REQUESTED DATA FROM 16 ANSWERS -
CONTINUE? Y/(N):y


L5  ANSWER 1 OF 16  CAPLUS  COPYRIGHT 2000 ACS
AN  1999:763123  CAPLUS
DN  132:44343
TI  Apoptosis regulating proteins as targets of therapy for
hematological
    malignancies
AU  Kornblau, Steven M.; Konopleva, Marina; Andreeff, Michael
CS  Department of Blood and Marrow Transplantation, Section of
Molecular
    Hematology and Therapy, The University of Texas M. D. Anderson
Cancer
    Center, Houston, TX, USA
SO  Expert Opin. Invest. Drugs (1999), 8(12), 2027-2057
    CODEN: EOIDER; ISSN: 1354-3784
PB  Ashley Publications
DT  Journal;  ***General Review***
LA  English
AB  A review with 306 refs. Most chemotherapeutic agents used in
the
    treatment of haematol. malignancies cause cell death by inducing
apoptosis
    through undefined means. The discovery of the proteins involved
in
    apoptosis and the description of apoptotic pathways suggest new
potential
    targets for therapeutic intervention. Both "intrinsic" and "extrinsic"
    pathways can be activated sep., but activation of caspases appears
central
    to most apoptotic pathways. Novel approaches attempt to induce
apoptosis
    by directly targeting a portion of an apoptotic pathway. Agents that
    trigger signalling of Fas or  ***tumor***  ***necrosis***
     ***factor***  (TNF)-  ***related***  apoptosis inducing ligand
(TRAIL)
    receptor seek to induce the extrinsic pathway at the cell surface.
The
    BCL-2  ***family***  of proteins seems central to the regulation
of
    those apoptotic pathways that involve mitochondrial sequestration
or the
    release of cytochrome c, with subsequent activation of Apaf-1,
caspase-9
    and caspase-3. The activity of this  ***family***  may depend
upon both
    the phosphorylation state of different members and the relative
level of
    pro- and anti-apoptotic members. New agents such as the
staurosporine
    analog UCN-01 and bryostatin are thought to affect apoptosis
induction by
    altering BCL-2 phosphorylation. Others, such as BCL-2 antisense
and ATRA
    attempt to modulate the protein levels to promote apoptosis. Direct
    activation of caspase-3 is a probable target, but as yet no agent with
    this direct  ***function***  is in trial. Clin. trials of several
    agents have been completed or are underway. It is likely that
agents that
    target particular points in apoptosis pathways will have
    antileukemia/lymphoma activity, however, the optimal utilization
may
    involve combination with other more conventional agents that also
activate
    apoptosis.
RE.CNT  307
RE
(1) Adida, C; Am J Pathol 1998, V152, P43 CAPLUS
(3) Akiyama, T; Anticancer Res 1999, V10, P67 CAPLUS
(4) Akiyama, T; Cancer Res 1997, V57, P1495 CAPLUS
(5) Alnemri, E; Proc Natl Acad Sci USA 1992, V89, P7295 CAPLUS
(6) Altieri, D; FASEB J 1995, V9, P860 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5  ANSWER 2 OF 16  CAPLUS  COPYRIGHT 2000 ACS
AN  1999:744668  CAPLUS
DN  132:235500
TI  ***Tumor***  ***necrosis***  ***factor***  receptors in
systemic
```

inflammation
AU Lin, E.; Calvano, S. E.; Lowry, S. F.
CS Department of Surgery, New York Hospital, Queens Flushing, NY, USA
SO Update Intensive Care Emerg. Med. (2000), 31(Immune Response in the
Critically Ill), 365-384
CODEN: UICMFN; ISSN: 0933-6788
PB Springer-Verlag
DT Journal; ***General Review***
LA English
AB A review with 114 refs. of what is known about ***tumor*** ***necrosis*** ***factor*** (TNF) receptor ***function*** and
signal transduction as they relate to inflammation. Due to similarities
in receptor structure and signaling pathways, the ***function*** of
related TNF receptor ***family*** members during systemic
inflammation is also highlighted. Finally, the authors focus on
clin.-derived data, beginning with immunocyte TNF receptor alterations
exhibited during acute systemic inflammation and culminating in potential
clin. implications stemming from such changes.
RE.CNT 114
RE
(1) Abraham, E; JAMA 1997, V277, P1531 CAPLUS
(2) Adam, D; Biochem J 1998, V333, P343 CAPLUS
(3) Aderka, D; J Exp Med 1992, V175, P323 CAPLUS
(4) Anrather, J; J Clin Invest 1997, V99, P763 CAPLUS
(8) Bazzoni, F; N Engl J Med 1996, V334, P1717 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:664951 CAPLUS
DN 132:11436
TI Interleukin-18
AU Dinarello, Charles A.
CS Department of Medicine, Division of Infectious Diseases, B168, University
of Colorado Health Sciences Center, Denver, CO, 80262, USA
SO Methods (Orlando, Fla.) (1999), 19(1), 121-132
CODEN: MTHDE9; ISSN: 1046-2023
PB Academic Press
DT Journal; ***General Review***
LA English
AB A review with 81 refs. summarizing the present knowledge on IL-18, to give
an insight into the future perspectives for its possible use as vaccine
adjuvant. Formerly called interferon (IFN) .gamma. inducing factor
(IGIF), IL-18 is the new name of a novel cytokine that plays an important
role in the T-helper type 1 (Th1) response, primarily by its ability to
induce IFN.gamma. prodn. in T cells and natural killer (NK) cells. IL-18
is ***related*** to the IL-1 ***family*** in terms of structure,
receptor ***family*** , and ***function*** . Also similar to
IL-1.beta., IL-18 is synthesized as a biol. inactive precursor mol.
lacking a signal peptide which requires cleavage into an active, mature
mol. by the intracellular cysteine protease called IL-1.beta.-converting
enzyme (ICE, also called caspase-1). The activity of mature IL-18 is
closely ***related*** to that of IL-1. IL-18 induces gene expression
and synthesis of ***tumor*** ***necrosis*** ***factor*** (TNF), IL-1, Fas ligand, and several chemokines. The activity of IL-18 is
via an IL-18 receptor (IL-18R) complex. This IL-18R complex is made up of
a binding chain termed IL-18R.alpha., a member of the IL-1 receptor
family previously identified as the IL-1 receptor-***related***
protein (IL-1Rrp), and a signaling chain, also a member of the IL-1R
family . The IL-18R complex recruits the IL-1R-activating kinase
(IRAK) and TNFR-assocd. factor-6 (TRAF-6) which phosphorylates nuclear
factor .kappa.B (NF.kappa.B)-inducing kinase (NIK) with subsequent
activation of NF.kappa.B. Thus, on the basis of primary structure,
3-dimensional structure, receptor ***family*** , signal transduction
pathways, and biol. effects, IL-18 appears to be a new member of the IL-1
family . (c) 1999 Academic Press.
RE.CNT 81
RE
(1) Adachi, O; Immunity 1998, V9, P143 CAPLUS
(2) Barbulescu, K; J Immunol 1998, V160, P3642 CAPLUS
(3) Bohn, E; J Immunol 1998, V160, P299 CAPLUS
(4) Boraschi, D; Eur Cytokine Netw 1998, V9, P205 CAPLUS
(5) Born, T; J Biol Chem 1998, V273, P29445 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:641810 CAPLUS
DN 131:349988
TI The ***tumor*** ***necrosis*** ***factor*** (TNF) ***family*** and ***related*** molecules
AU Wallach, David; Bigda, Jacek; Engelmann, Hartmut
CS Department of Biological Chemistry, The Weizmann Institute of Science,
Rehovot, 76100, Israel
SO Cytokine Network Immune Funct. (1999), 51-84. Editor(s): Theze, Jacques.
Publisher: Oxford University Press, Oxford, UK.
CODEN: 68GGAA
DT Conference; ***General Review***
LA English
AB A review with 31 refs. Topics discussed include common features of
family members; occurrence of ligands and receptors; common and
distinct effects of the TNF ligand and receptor families; cellular origins
of TNFs and their receptors; ***functions*** of TNFs; structure-
function relationships in TNFs and their receptors; intracellular
domains of TNF receptors; HVEM and LIGHT; CD95; Apo-3 and Apo3-L; TRAIL;
CAR1; Osteoprotegerin; TRANCE; RANK; CD40; CD40-L; GITR; OX40; TACI; and
APRIL.
RE.CNT 10
RE
(1) Beutler, B; Science 1994, V264, P667 CAPLUS
(2) Cosman, D; Stem Cells 1994, V12, P440 CAPLUS
(4) Gruss, H; Blood 1995, V85, P3378 CAPLUS
(5) Meakin, S; Trends Neurosci 1992, V15, P323 CAPLUS
(7) Smith, C; Cell 1994, V76, P959 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:636341 CAPLUS
DN 131:226786
TI Aberrant apoptotic signals in tumorigenesis
AU Sakamuro, Daitoku
CS Walther Cancer Inst. Dep. Med. Chem. Mol. Pharmacol., Purdue Univ. Cancer
Cent., USA
SO Jikken Igaku (1999), 17(14), 1911-1918
CODEN: JIIGEF; ISSN: 0288-5514
PB Yodosha
DT Journal; ***General Review***
LA Japanese
AB A review with 41 refs., on (1) p53 activation and apoptosis induced by DNA
damage, (2) p53-mediated apoptosis induced by oxidative stress, (3) roles
of p19ARF, BIN1, Fas/Fas ligand, and cytochrome c in c-myc-dependent
apoptosis and tumorigenesis, (4) structure and pathophysiol.
functions of TNF receptor ***family*** mols. in apoptosis, and
(5) possible use of TRAIL (TNF- ***related*** apoptosis-inducing
ligand) in cancer treatment.

L5 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:404130 CAPLUS
DN 131:183537

TI TRANCE is a TNF ***family*** member that regulates dendritic cell and
osteoclast ***function***
AU Wong, Brian R.; Josien, Regis; Choi, Yongwon
CS Laboratory of Immunology, The Rockefeller University, New York, NY, 10021,
USA
SO J. Leukocyte Biol. (1999), 65(6), 715-724
CODEN: JLBIE7; ISSN: 0741-5400
PB Federation of American Societies for Experimental Biology
DT Journal; ***General Review***
LA English
AB A review with 75 refs. ***Tumor*** ***necrosis*** ***factor***
(TNF)- ***related*** activation-induced cytokine (TRANCE) is a new
member of the TNF ***family*** emerging as a key regulator of the
immune system and of bone development and homeostasis. TRANCE is
expressed on activated T cells and activates mature dendritic cells (DC),
suggesting that it plays a role in the T cell-DC interaction during an
immune response. Furthermore, TRANCE is expressed on osteoblasts
stimulated with vitamin D3, dexamethasone, and parathyroid hormone.
TRANCE, when expressed on osteoblasts, induces osteoclastogenesis and
osteoclast activation, suggesting that it links known calciotropic
hormones to bone resorption. TRANCE mediates its effects via the
TRANCE-receptor (TRANCE-R/RANK), whereas its activity can be inhibited by
the sol. decoy receptor osteoprotegerin/osteoclast inhibitory factor
(OPG/OCIF). OPG can be neutralized by another TNF-***family***
member, the TNF- ***related*** apoptosis-inducing ligand (TRAIL),
suggesting that TRANCE is part of a complex cytokine network that
regulates a diverse set of ***functions*** . The authors discuss the
current literature describing TRANCE and its receptors and its role in
controlling DC and osteoclast ***function*** .
RE.CNT 75
RE
(1) Anderson, D; Nature 1997, V390, P175 CAPLUS
(2) Baeuerle, P; Science 1988, V242, P540 CAPLUS
(3) Banchereau, J; Nature 1998, V392, P245 CAPLUS
(4) Bennett, S; Nature 1998, V393, P478 CAPLUS
(5) Bucay, N; Genes Dev 1998, V12, P1260 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:315231 CAPLUS
DN 131:100943
TI To die or not to die-the quest of the TRAIL receptors
AU Degli-Esposti, Mariapia
CS Department of Microbiology, QEII Medical Centre, The University of Western
Australia, Perth, 6009, Australia
SO J. Leukocyte Biol. (1999), 65(5), 535-542
CODEN: JLBIE7; ISSN: 0741-5400
PB Federation of American Societies for Experimental Biology
DT Journal; ***General Review***
LA English
AB A review with 59 refs. The last 18 mo have witnessed the characterization
of several new members of the ***tumor*** ***necrosis*** ***factor*** (TNF) receptor ***family*** . Among these are five
receptors for the cytotoxic ligand TRAIL (TNF- ***related*** apoptosis-inducing ligand). Two of these receptors, TRAIL-R1 and
TRAIL-R2, contain classical cytoplasmic death domains and are able to
transduce an apoptotic signal. The others lack functional death domains
and are not able to promote cell death. Indeed, one of the receptors for
TRAIL, osteoprotegerin (OPG) is a sol. protein whose activities so far
have been shown to be inhibition of osteoclastogenesis and increased bone
d. in vivo. The existence of multiple receptors for TRAIL suggests an
unexpected complexity to TRAIL-mediated biol. ***functions*** .
RE.CNT 59
RE
(1) Amakawa, R; Cell 1996, V84, P551 CAPLUS
(2) Anderson, D; Nature 1997, V390, P175 CAPLUS
(5) Bodmer, J; Immunity 1997, V6, P79 CAPLUS
(6) Browning, J; J Exp Med 1996, V183, P867 CAPLUS
(8) Chicheportiche, Y; J Biol Chem 1997, V272, P32401 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:182258 CAPLUS
DN 131:27970
TI A New Member of ***Tumor*** ***Necrosis*** ***Factor*** Ligand
Family , ODF/OPGL/TRANCE/RANKL, Regulates Osteoclast
Differentiation and ***Function***
AU Takahashi, Naoyuki; Udagawa, Nobuyuki; Suda, Tatsuo
CS Department of Biochemistry, School of Dentistry, Showa University, Tokyo,
142-8555, Japan
SO Biochem. Biophys. Res. Commun. (1999), 256(3), 449-455
CODEN: BBRCA9; ISSN: 0006-291X
PB Academic Press
DT Journal; ***General Review***
LA English
AB A review and discussion with 55 refs. Osteoclasts, the multinucleated
giant cells that resorb bone, develop from monocyte-macrophage lineage
cells. Osteoblasts or bone marrow stromal cells have been suggested to be
involved in osteoclastic bone resorption. The recent discovery of new
members of the ***tumor*** ***necrosis*** ***factor*** (TNF)
receptor-ligand ***family*** has elucidated the precise mechanism by
which osteoblasts/stromal cells regulate osteoclast differentiation and
function . Osteoblasts/stromal cells express a new member of the
TNF-ligand ***family*** 'osteoclast differentiation
factor(ODF)/osteoprotegerin ligand (OPGL)/TNF- ***related*** activation-induced cytokine (TRANCE)/receptor activator of NF-kB ligand
(RANKL)' as a membrane assocd. factor. Osteoclast precursors which
possess RANK, a TNF receptor ***family*** member, recognize
ODF/OPGL/TRANCE/RANKL through cell-to-cell interaction with
osteoblasts/stromal cells, and differentiate into osteoclasts in the
presence of macrophage colony-stimulating factor. Mature osteoclasts also
express RANK, and their bone-resorbing activity is also induced by
ODF/OPGL/TRANCE/RANKL which osteoblasts/stromal cells possess.
Osteoprotegerin (OPG)/osteoclastogenesis inhibitory factor (OCIF)/TNF
receptor-like mol. 1 (TR1) is a sol. decoy receptor for ODF/OPGL/
TRANCE/RANKL. Activation of NF-kB and c-Jun N-terminal kinase through the
RANK-mediated signaling system appears to be involved in differentiation
and activation of osteoclasts. (c) 1999 Academic Press.
RE.CNT 55
RE
(1) Akatsu, T; Bone 1998, V23, P495 CAPLUS
(2) Anderson, D; Nature 1997, V390, P175 CAPLUS
(5) Brandstrom, H; Biochem Biophys Res Commun 1998, V248, P454 CAPLUS
(6) Bucay, N; Genes Dev 1998, V12, P1260 CAPLUS
(7) Chambers, T; Proc Natl Acad Sci USA 1993, V90, P5578 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:156605 CAPLUS
DN 130:178865
TI Structural biology of apoptosis proteins. Recent advances in structural

analysis of TNF- ***related*** , Fas- ***related*** , Bcl-2 ***family*** and caspase ***family*** proteins
AU Aritomi, Masaharu; Ohta, Shigeo
CS Dep. Struct. Biol., Biomol. Eng. Res. Inst., Suita, 565-0874, Japan
SO Tanpakushitsu Kakusan Koso (1999), 44(4), 395-403
CODEN: TAKKAJ; ISSN: 0039-9450
PB Kyoritsu Shuppan
DT Journal; ***General Review***
LA Japanese
AB A review with 25 refs., on (1) transduction of apoptotic signals in Caenorhabditis elegans, (2) TNF- or Fas ligand-mediated signal transduction in mammals, (3) mitochondria-mediated signal transduction of apoptosis, (4) conformation of TNF and its receptor, (5) three-dimensional structure of Fas death domain and Fas-assocd. protein sith death domain (FADD), (6)structure and ***function*** of Bcl-2 ***family*** proteins, an d(7) crystal structure of caspase ***family*** proteins.

L5 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1998:761671 CAPLUS
DN 130:134227
TI Control of neuronal survival by neurotrophins
AU Frade, Jose Maria; Casademunt, Elisabeth; Dechant, Georg; Barde, Yves-Alain
CS Max Planck Inst. Psychiatry, Planegg-Martinsried, Germany
SO Verh. - K. Ned. Akad. Wet., Afd. Natuurkd., Tweede Reeks (1998), 100(Pharmaceutical Intervention in Apoptotic Pathways), 87-96
CODEN: VNAWAG; ISSN: 0373-465X
PB North-Holland
DT Journal; ***General Review***
LA English
AB A review with 59 refs. Neurotrophins are ***related*** secretory proteins that control cell survival in the nervous system. All can prevent programmed cell death by binding to specific cell surface receptors belonging to a ***family*** of tyrosine kinase receptors. As these receptors are expressed in subgroups of developing neurons, interference with the ***function*** of these receptors or of their ligands leads to selective neuronal deficits in the nervous system. All neurotrophins also bind to another receptor designated the neurotrophin receptor p75. This member of the ***tumor*** ***necrosis*** ***factor*** receptor ***family*** can be activated by nerve growth factor, leading to the death of neurons in the developing nervous system. Thus, the neurotrophin nerve growth factor controls cell nos. in opposite ways by its ability to activate 2 different receptors.
RE.CNT 60
RE
(1) Barbacid, M; J Neurobiol 1994, V25, P1386 CAPLUS
(2) Bothwell, M; Annu Rev Neurosci 1995, V18, P223 CAPLUS
(3) Bovolenta, P; J Neuroscience 1996, V16, P4402 CAPLUS
(4) Carter, B; Science 1996, V272, P542 CAPLUS
(5) Casaccia-Bonnefil, P; Nature 1996, V383, P716 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1998:375207 CAPLUS
DN 129:117886
TI Neurotrophins: the biological paradox of survival factors eliciting apoptosis
AU Casaccia-Bonnefil, Patrizia; Kong, Haeyoung; Chao, Moses V.
CS Molecular Neurobiology Program, Skirball Institute, NY, 10016, USA
SO Cell Death Differ. (1998), 5(5), 357-364
CODEN: CDDIEK; ISSN: 1350-9047
PB Stockton Press
DT Journal; ***General Review***
LA English
AB A review with .apprx.50 refs. Neurotrophins are target-derived sol. polypeptides required for neuronal survival. Binding of neurotrophins to Trk receptor tyrosine kinases initiate signaling cascades that promote cell survival and differentiation. All ***family*** members bind to another receptor (p75NTR), which belongs to the ***tumor*** ***necrosis*** ***factor*** superfamily. Hence, nerve growth factor (NGF) and ***related*** trophic factors are unique in that two sep. receptor types are utilized. Although the biol. ***function*** of p75NTR has been elusive, it has been suggested to mediate apoptosis of developing neurons in the absence of Trk receptors. This presents a tantalizing paradigm, in which life-death decisions of cells are dependent upon the expression and action of two different receptors with distinctive signaling mechanisms. In the presence of TrkA receptors, p75 can participate in the formation of high affinity binding sites and enhanced NGF responsiveness leading to a survival signal. In the absence of TrkA receptors, p75 can generate, in only specific cell populations, a death signal. Here we discuss the unique features and implications of this unusual signal transduction system.

L5 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1998:292210 CAPLUS
DN 129:94051
TI The TRAIL of death
AU Goodwin, R. G.; Smith, C. A.
CS Immunex Corporation, Seattle, WA, 98101, USA
SO Apoptosis (1998), 3(2), 83-88
CODEN: APOPFN; ISSN: 1360-8185
PB Rapid Science Ltd.
DT Journal; ***General Review***
LA English
AB A review with 44 refs. The TNF ligand ***family*** member termed TRAIL has been shown to induce apoptosis in a wide variety of transformed cell lines. The normal ***functions*** of this cytokine in vivo remain, however, relatively unknown. The complexity of this biol. system has now increased unexpectedly with the identification of four distinct receptors for TRAIL, two of which have cytoplasmic death domains. This review will describe the known biol. effects of TRAIL, as well as the structure and possible ***functions*** of its recently identified receptors.

L5 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1997:749108 CAPLUS
DN 128:43897
TI Eph ***family*** receptors and ligands in vascular cell targeting and assembly
AU Stein, Elke; Schoecklmann, Harald; Daniel, Thomas O.
CS Department of Pharmacology, Vanderbilt University Medical Center, Nashville, TN, USA
SO Trends Cardiovasc. Med. (1997), 7(8), 329-334
CODEN: TCMDEQ; ISSN: 1050-1738
PB Elsevier
DT Journal; ***General Review***
LA English
AB A review, with 52 refs. Members of the Eph ***family*** of receptor tyrosine kinases det. neural cell aggregation and targeting behavior, ***functions*** that are also crit. in vascular assembly and remodeling. Among this class of diverse receptors EphA2 (Eck) and EphB1 (ELK) represent prototypes for two receptor subfamilies distinguished by high-affinity interaction with either glycerophosphatidylinositol (GPI)-linked or transmembrane ligands, resp. EphA2 participates in angiogenic responses to ***tumor*** ***necrosis*** ***factor*** (TNF) through an autocrine loop affecting endothelial cell

migration EphB1
and its ligand Ephrin-B1 (LERK-2) are important determinants of assembly
of endothelial cells from the microvasculature of the kidney where both
are expressed in endothelial progenitors and in glomerular microvascular
endothelial cells. Ephrin-B1 activation of EphB1 promotes assembly of
these cells into capillary-like structures. Interaction trap approaches
have identified downstream signaling proteins that complex with
ligand-activated EphA2 or EphB1, including nonreceptor tyrosine kinase and
SH2 domain-contg. adapter proteins. The Grb 10 adapter is one of a subset
that binds activated EphB1, but not EphA2, defining distinct signaling
mechanisms for these ***related*** endothelial receptors. On the
basis of observations in vascular endothelial cells and recent results
defining, Eph receptor and ligand roles in neural cell targeting, we
propose that these receptors direct cell-cell recognition events that are
crit. in vasculogenesis and angiogenesis.

L5 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1996:624415 CAPLUS
DN 125:272074
TI Common aspects of the signal transduction mechanism of the Epstein-Barr
virus (EBV) transforming protein latent membrane protein LMP1 and members
of TNF receptor ***family***
AU Harada, Shizuko; Mosialos, George
CS Department of Microbiology and Molecular Genetics, Harvard Medical School,
Boston, MA, USA
SO Saibo Kogaku (1996), 15(9), 1241-1248
CODEN: SAKOEO; ISSN: 0287-3796
DT Journal; ***General Review***
LA Japanese
AB A review with 32 refs., on LMP1 and malignant tumor, structure and
function of LMP1, recombinant EBV expts., investigation of LMP1
binding protein, structure and ***function*** of TNF receptor assocd.
factor (TRAF), and LMP1 and TRAF- ***related*** transformation model.

L5 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1992:529436 CAPLUS
DN 117:129436
TI Interleukin-8, a chemotactic and inflammatory cytokine
AU Baggiolini, Marco; Clark-Lewis, Ian
CS Theodor-Kocher Inst., Univ. Bern, Bern, CH-3000, Switz.
SO FEBS Lett. (1992), 307(1), 97-101
CODEN: FEBLAL; ISSN: 0014-5793
DT Journal; ***General Review***
LA English
AB A review with 38 refs. Interleukin-8 (IL-8) belongs to a ***family***
of small, structurally ***related*** cytokines similar to platelet
factor 4. It is produced by phagocytes and mesenchymal cells exposed to
inflammatory stimuli (e.g., interleukin-1 or ***tumor*** ***necrosis*** ***factor***) and activates neutrophils inducing
chemotaxis, exocytosis and the respiratory burst. In vivo, IL-8 elicits a
massive neutrophil accumulation at the site of injection. Five
neutrophil-activating cytokines similar to IL-8 in structure and
function have been identified recently. IL-8 and the
related cytokines are produced in several tissues upon infection,
inflammation, ischemia, trauma etc., and are thought to be the main cause
of local neutrophil accumulation.

L5 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1991:623497 CAPLUS
DN 115:223497
TI A new superfamily of cell surface proteins ***related*** to the nerve
growth factor receptor
AU Mallett, Susan; Barclay, A. Neil
CS Sir William Dunn Sch. Pathol., Univ. Oxford, Oxford, UK
SO Immunol. Today (1991), 12(7), 220-3
CODEN: IMTOD8; ISSN: 0167-4919
DT Journal; ***General Review***
LA English
AB A review, with 33 refs., of the mol. functional features of the title
proteins. These include 2 lymphocyte proteins of unknown ***function***
and 2 receptors for ***tumor*** ***necrosis*** ***factor*** .
These are cysteine-rich membrane proteins and probably ***function***
as receptors for cytokines.

=> d his

(FILE 'HOME' ENTERED AT 11:21:22 ON 10 JUL 2000)

FILE 'CAPLUS' ENTERED AT 11:21:27 ON 10 JUL 2000
L1 1390059 S REVIEW/DT
L2 2613 S L1 AND (TUMOR OR TUMOUR)(W)(NECROSIS FACTOR)/AB,BI
L3 206 S L2 AND RELATED/AB,BI
L4 42 S L3 AND FAMILY/AB,BI
L5 16 S L4 AND FUNCTION#/AB,BI
L6 0 S L5 AND HOMOLOGY/AB,BI

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		56.09	56.24

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

	ENTRY	SINCE FILE SESSION	TOTAL
CA SUBSCRIBER PRICE		-8.90	-8.90

STN INTERNATIONAL LOGOFF AT 11:23:19 ON 10 JUL 2000

L4 ANSWER 5 OF 11 MEDLINE
AN 1999175482 MEDLINE
DN 99175482
TI Identification of a new member of the **tumor necrosis factor family** and its receptor, a human ortholog of mouse GITR.
AU Gurney A L; Marsters S A; Huang R M; Pitti R M; Mark D T; Baldwin D T; Gray A M; Dowd A D; Brush A D; Heldens A D; Schow A D; Goddard A D; Wood W I; Baker K P; Godowski P J; Ashkenazi A
CS Department of Molecular Biology Genentech Inc. 1 DNA Way South San Francisco California 94080 USA.
SO CURRENT BIOLOGY, (1999 Feb 25) 9 (4) 215-8.
Journal code: B44. ISSN: 0960-9822.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
EW 19990604
AB The tumor necrosis factor (TNF) and TNF receptor (TNFR) gene superfamilies regulate diverse biological functions, including cell proliferation, differentiation, and survival [1] [2] [3]. We have identified a new TNF-**related** ligand, designated human GITR ligand (hGITRL), and its human receptor (hGITR), an ortholog of the recently discovered murine glucocorticoid-induced TNFR-**related** (mGITR) protein [4]. The hGITRL gene mapped to chromosome 1q23, near the gene for the TNF homolog Fas/CD95 ligand [5]. The hGITR gene mapped to chromosome 1p36, near a cluster of five genes encoding TNFR homologs [1] [6]. We found hGITRL mRNA in several peripheral tissues, and detected hGITRL protein on cultured vascular endothelial cells. The levels of hGITR mRNA in tissues were generally low; in peripheral blood T cells, however, antigen-receptor stimulation led to a substantial induction of hGITR transcripts. Cotransfection of hGITRL and hGITR in embryonic kidney 293 cells activated the anti-apoptotic transcription factor NF-kappaB, via a pathway that appeared to involve TNFR-associated factor 2 (TRAF2) [7] and NF-kappaB-inducing kinase (NIK) [8]. Cotransfection of hGITRL and hGITR in Jurkat T leukemia cells inhibited antigen-receptor-induced cell death. Thus, hGITRL and hGITR may modulate T lymphocyte survival in peripheral tissues.

```
**********STN Columbus **********
**

FILE 'HOME' ENTERED AT 10:33:47 ON 10 JUL 2000

=> file medline

COST IN U.S. DOLLARS                SINCE FILE     TOTAL
                                    ENTRY   SESSION
FULL ESTIMATED COST                   0.15      0.15

FILE 'MEDLINE' ENTERED AT 10:33:58 ON 10 JUL 2000

 FILE LAST UPDATED: 6 JUL 2000 (20000706/UP). FILE
COVERS 1960 TO DATE.

 MEDLINE has been reloaded to reflect the annual MeSH changes
made by
 the National Library of Medicine for 2000. Enter HELP RLOAD for
details.

 OLDMEDLINE, data from 1960 through 1965 from the Cumulated
Index
 Medicus (CIM), has been added to MEDLINE. See HELP
CONTENT for details.

 Left, right, and simultaneous left and right truncation are available in
the
 Basic Index. See HELP SFIELDS for details.

 THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY
AND ACCURATE
 SUBSTANCE IDENTIFICATION.

=> s trell/ab,bi

        1 TRELL/BI
  5348796 AB/FA
        1 TRELL/AB
          (TRELL/BI (L) AB/FA)
        1 TRELL/BI
L1      1 TRELL/AB,BI

=> d bib ab


L1  ANSWER 1 OF 1  MEDLINE
AN  76058643    MEDLINE
DN  76058643
TI  Hydantoin derivatives and malignancies of the haemopoietic
system.
AU  Bichel J
SO  ACTA MEDICA SCANDINAVICA, (1975 Oct) 198 (4) 327-8.
    Journal code: 14G. ISSN: 0001-6101.
CY  Sweden
DT  Journal; Article; (JOURNAL ARTICLE)
LA  English
FS  Abridged Index Medicus Journals; Priority Journals
EM  197603
AB  Two patients are described who developed malignant lymphoma
    (lymphosarcoma) after diphenylydantoin therapy because of
epilepsy.
    Malignant lymphoma in a few patients receiving this medication
has been
    described earlier. The literature has been reviewed and discussed
recently
    by Rausing and  ***Trell***  (2).


=> s tumor necrosis factor family related protein#/ab,bi

   402104 TUMOR/BI
   106882 NECROSIS/BI
   444330 FACTOR/BI
   244109 FAMILY/BI
   561805 RELATED/BI
  1183373 PROTEIN#/BI
  5348796 AB/FA
        0 TUMOR NECROSIS FACTOR FAMILY RELATED
PROTEIN#/AB

((TUMOR(W)NECROSIS(W)FACTOR(W)FAMILY(W)RELATE
D(W)PROTEIN#)/BI
          (L) AB/FA)
   402104 TUMOR/BI
   106882 NECROSIS/BI
   444330 FACTOR/BI
   244109 FAMILY/BI
   561805 RELATED/BI
  1183373 PROTEIN#/BI
        0 TUMOR NECROSIS FACTOR FAMILY RELATED
PROTEIN#/BI

((TUMOR(W)NECROSIS(W)FACTOR(W)FAMILY(W)RELATE
D(W)PROTEIN#)/BI)
L2      0 TUMOR NECROSIS FACTOR FAMILY RELATED
PROTEIN#/AB,BI

=> s tumor necrosis factor family/ab,bi

   402104 TUMOR/BI
   106882 NECROSIS/BI
   444330 FACTOR/BI
   244109 FAMILY/BI
  5348796 AB/FA
       40 TUMOR NECROSIS FACTOR FAMILY/AB
          ((TUMOR(W)NECROSIS(W)FACTOR(W)FAMILY)/BI
(L) AB/FA)
   402104 TUMOR/BI
   106882 NECROSIS/BI
   444330 FACTOR/BI
   244109 FAMILY/BI
       53 TUMOR NECROSIS FACTOR FAMILY/BI

((TUMOR(W)NECROSIS(W)FACTOR(W)FAMILY)/BI)
L3      53 TUMOR NECROSIS FACTOR FAMILY/AB,BI

=> s l3 and related/ab,bi

   561805 RELATED/BI
  5348796 AB/FA
   454744 RELATED/AB
          (RELATED/BI (L) AB/FA)
   561805 RELATED/BI
L4      11 L3 AND RELATED/AB,BI

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 11 ANSWERS -
CONTINUE? Y/(N):y


L4  ANSWER 1 OF 11  MEDLINE
AN  2000219071    MEDLINE
DN  20219071
TI  Prognostic relevance of altered Fas (CD95)-system in human
breast cancer.
AU  Mottolese M; Buglioni S; Bracalenti C; Cardarelli M A;
Ciabocco L;
    Giannarelli D; Botti C; Natali P G; Concetti A; Venanzi F M
CS  Pathology Department, Regina Elena Cancer Institute, Rome,
Italy.
SO  INTERNATIONAL JOURNAL OF CANCER, (2000 Mar 20)
89 (2) 127-32.
    Journal code: GQU. ISSN: 0020-7136.
CY  United States
DT  Journal; Article; (JOURNAL ARTICLE)
LA  English
FS  Priority Journals; Cancer Journals
EM  200006
EW  20000605
AB  The Fas ligand (FasL) and its receptor Fas (APO-1 or CD95) are
members,
    respectively, of the  ***tumor***  ***necrosis***
***factor***
     ***family***  that, upon interaction with each other, play a key
role in
    the initiation of one apoptotic pathway. Faulty regulation of the Fas
    system has been described in a variety of human tumors with
different
    histogenetic origin. Here, we describe the expression and
distribution of
    Fas receptor and ligand pair antigens in surgical samples collected
from a
    cohort of 186 patients bearing breast neoplasms (45 benign and 141
    malignant lesions). Immunoperoxidase staining of formalin-fixed
tissues
    showed that 91.1% of benign lesions expressed Fas, which was
present in
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only 56.7% of malignant tumors. On the other hand, FasL was found positive in 22.2% of benign neoplasms and up-regulated in in situ as well as invasive carcinomas (53.9%). Moreover, in malignant tumors, the expression of receptor and ligand antigens appeared to be inversely ***related*** . When these findings were correlated with pathological parameters of prognostic relevance, a significant association was observed between FasL and the presence of metastatic lymph nodes and larger tumor size while Fas expression correlated to node-negative status and smaller tumor size.

Patients with Fas positive tumors exhibited longer disease-free survival than those with Fas-negative carcinoma while FasL did not influence patient outcome. These relationships indicate that benign and malignant mammary lesions are characterized by differential cellular expression of Fas and FasL and suggest that a neoplastic Fas negative/FasL positive phenotype may be linked to breast cancer progression. Copyright 2000 Wiley-Liss, Inc.

L4 ANSWER 2 OF 11 MEDLINE
AN 2000130293 MEDLINE
DN 20130293
TI TRANCE, a ***tumor*** ***necrosis*** ***factor*** ***family*** member, enhances the longevity and adjuvant properties of dendritic cells in vivo.
AU Josien R; Li H L; Ingulli E; Sarma S; Wong B R; Vologodskaia M; Steinman R M; Choi Y
CS Laboratory of Cellular Physiology and Immunology, The Rockefeller University, New York, New York 10021, USA.
NC AI13013 (NIAID)
AI44264 (NIAID)
DK39672 (NIDDK)
SO JOURNAL OF EXPERIMENTAL MEDICINE, (2000 Feb 7) 191 (3) 495-502.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Cancer Journals; Priority Journals
EM 200006
AB Mature dendritic cells (DCs) are powerful antigen presenting cells that have the unique capacity to migrate to the T cell zone of draining lymph nodes after subcutaneous injection. Here we report that treatment of antigen-pulsed mature DCs with tumor necrosis factor (TNF)-***related*** activation-induced cytokine (TRANCE), a TNF family member, before immunization enhances their adjuvant capacity and elicits improved T cell priming in vivo, such that both primary and memory T cell immune responses are enhanced. By enumerating migratory DCs in the draining lymph nodes and by studying their function in stimulating naive T cells, we show that one of the underlying mechanisms for enhanced T cell responses is an increase in the number of ex vivo antigen-pulsed DCs that are found in the T cell areas of lymph nodes. These results suggest that the longevity and abundance of mature DCs at the site of T cell priming influence the strength of the DC-initiated T cell immunity in situ. Our findings have the potential to improve DC-based immunotherapy; i.e., the active immunization of humans with autologous DCs that have been pulsed with clinically significant antigens ex vivo.

L4 ANSWER 3 OF 11 MEDLINE
AN 1999290669 MEDLINE
DN 99290669
TI Relation of TNF- ***related*** apoptosis-inducing ligand (TRAIL) receptor and FLICE-inhibitory protein expression to TRAIL-induced apoptosis of melanoma.
AU Zhang X D; Franco A; Myers K; Gray C; Nguyen T; Hersey P
CS Immunology and Oncology Unit, Department of Surgical Sciences, Newcastle, NSW, Australia.
SO CANCER RESEARCH, (1999 Jun 1) 59 (11) 2747-53.
Journal code: CNF. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199909
EW 19990901
AB Past studies have shown that apoptosis mediated by TNF-***related*** apoptosis-inducing ligand (TRAIL) is regulated by the expression of two death receptors [TRAIL receptor 1 (TRAIL-R1) and TRAIL-R2] and two decoy receptors (TRAIL-R3 and TRAIL-R4) that inhibit apoptosis. In previous studies, we have shown that TRAIL but not other members of the ***tumor*** ***necrosis*** ***factor*** ***family*** induce apoptosis in approximately two-thirds of melanoma cell lines. Here, we examined whether the expression of TRAIL-R at the mRNA and protein level in a panel of 28 melanoma cell lines and melanocytes correlated with their sensitivity to TRAIL-induced apoptosis. We report that at least three factors appear to underlie the variability in TRAIL-induced apoptosis. (a) Four of nine cell lines that were insensitive to TRAIL-induced apoptosis failed to express death receptors, and in two instances, lines were devoid of all TRAIL-Rs. Southern analysis suggested this was due to loss of the genes for the death receptors. (b) Despite the presence of mRNA for the TRAIL-R, some of the lines failed to express TRAIL-R protein on their surface. This was evident for TRAIL-R1 and more so for the TRAIL decoy receptors TRAIL-R3 and -R4. Studies on permeabilized cells revealed that the receptors were located within the cytoplasm and redistribution from the cytoplasm may represent a posttranslational control mechanism. (c) Surface expression of TRAIL-R1 and -R2 (but not TRAIL-R3 and -R4) showed an overall correlation with TRAIL-induced apoptosis. However, certain melanoma cell lines and clones were relatively resistant to TRAIL-induced apoptosis despite the absence of decoy receptors and moderate levels of TRAIL-R1 and -R2 expression. This may indicate the presence of inhibitors within the cells, but resistance to apoptosis could not be correlated with expression of the caspase inhibitor FLICE-inhibitory protein. mRNA for another TRAIL receptor, osteoprotegerin, was expressed in 22 of the melanoma lines but not on melanocytes. Its role in induction of apoptosis remains to be studied. These results appear to have important implications for future clinical studies on TRAIL.

L4 ANSWER 4 OF 11 MEDLINE
AN 1999207064 MEDLINE
DN 99207064
TI TRANCE, a ***tumor*** ***necrosis*** ***factor*** ***family*** member critical for CD40 ligand-independent T helper cell activation [see comments].

CM Comment in: J Exp Med 1999 Apr 5;189(7):1017-20
AU Bachmann M F; Wong B R; Josien R; Steinman R M; Oxenius A; Choi Y
CS Basel Institute for Immunology, CH 4005 Basel, Switzerland.
NC GM-07739 (NIGMS)
AI-44264 (NIAID)
AI-13013 (NIAID)
+
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1999 Apr 5) 189 (7) 1025-31.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199907
EW 19990702
AB CD40 ligand (CD40L), a tumor necrosis factor (TNF) family member, plays a
critical role in antigen-specific T cell responses in vivo. CD40L
expressed on activated CD4(+) T cells stimulates antigen-presenting cells
such as dendritic cells, resulting in the upregulation of costimulatory
molecules and the production of various inflammatory cytokines required
for CD4(+) T cell priming in vivo. However, CD40L- or CD40-deficient mice
challenged with viruses mount protective CD4(+) T cell responses that
produce normal levels of interferon gamma, suggesting a
CD40L/CD40-independent mechanism of CD4(+) T cell priming that to date has
not been elucidated. Here we show that CD4(+) T cell responses to viral
infection were greatly diminished in CD40-deficient mice by administration
of a soluble form of TNF- ***related*** activation-induced cytokine
receptor (TRANCE-R) to inhibit the function of another TNF family member,
TRANCE. Thus, the TRANCE/TRANCE-R interaction provides costimulation
required for efficient CD4(+) T cell priming during viral infection in the
absence of CD40L/CD40. These results also indicate that not even the
potent inflammatory microenvironment induced by viral infections is
sufficient to elicit efficient CD4(+) T cell priming without proper
costimulation provided by the TNF family (CD40L or TRANCE). Moreover, the
data suggest that TRANCE/TRANCE-R may be a novel and important target for
immune intervention.

L4 ANSWER 5 OF 11 MEDLINE
AN 1999175482 MEDLINE
DN 99175482
TI Identification of a new member of the ***tumor*** ***necrosis***
factor ***family*** and its receptor, a human ortholog of
mouse GITR.
AU Gurney A L; Marsters S A; Huang R M; Pitti R M; Mark D T; Baldwin D T;
Gray A M; Dowd A D; Brush A D; Heldens A D; Schow A D; Goddard A D; Wood W
I; Baker K P; Godowski P J; Ashkenazi A
CS Department of Molecular Biology Genentech Inc. 1 DNA Way South San
Francisco California 94080 USA.
SO CURRENT BIOLOGY, (1999 Feb 25) 9 (4) 215-8.
Journal code: B44. ISSN: 0960-9822.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
EW 19990604
AB The tumor necrosis factor (TNF) and TNF receptor (TNFR) gene superfamilies
regulate diverse biological functions, including cell proliferation,
differentiation, and survival [1] [2] [3]. We have identified a new TNF-
related ligand, designated human GITR ligand (hGITRL), and its
human receptor (hGITR), an ortholog of the recently discovered murine
glucocorticoid-induced TNFR- ***related*** (mGITR) protein [4]. The
hGITRL gene mapped to chromosome 1q23, near the gene for the TNF homolog
Fas/CD95 ligand [5]. The hGITR gene mapped to chromosome 1p36, near a
cluster of five genes encoding TNFR homologs [1] [6]. We found hGITRL mRNA
in several peripheral tissues, and detected hGITRL protein on cultured
vascular endothelial cells. The levels of hGITR mRNA in tissues were
generally low; in peripheral blood T cells, however, antigen-receptor
stimulation led to a substantial induction of hGITR transcripts.
Cotransfection of hGITRL and hGITR in embryonic kidney 293 cells activated
the anti-apoptotic transcription factor NF-kappaB, via a pathway that
appeared to involve TNFR-associated factor 2 (TRAF2) [7] and
NF-kappaB-inducing kinase (NIK) [8]. Cotransfection of hGITRL and hGITR in
Jurkat T leukemia cells inhibited antigen-receptor-induced cell death.
Thus, hGITRL and hGITR may modulate T lymphocyte survival in peripheral
tissues.

L4 ANSWER 6 OF 11 MEDLINE
AN 1999128078 MEDLINE
DN 99128078
TI Human astrocytic brain tumors express APO2L/TRAIL.
AU Rieger J; Ohgaki H; Kleihues P; Weller M
CS Department of Molecular Neurology, University of Tubingen, Germany.
SO ACTA NEUROPATHOLOGICA, (1999 Jan) 97 (1) 1-4.
Journal code: 1CE. ISSN: 0001-6322.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199908
EW 19990804
AB AP02 ligand (APO2L) is a CD95 ligand (CD95L)-***related*** cytokine of
the ***tumor*** ***necrosis*** ***factor*** ***family***
that interacts with agonistic (DR4, DR5) and antagonistic (DcR1, DcR2)
receptors. Cultured malignant glioma cells preferentially express
agonistic receptors and are susceptible to APO2L-induced apoptosis. Here,
we report that 8 of 8 human glioma cell lines expressed APO2L mRNA and
protein in vitro. Immunohistochemistry using a monoclonal antibody to
APO2L revealed that all 23 primary astrocytic brain tumors analyzed,
including low-grade astrocytomas and glioblastomas, express APO2L in vivo.
With the exception of reactive astrocytes, non-neoplastic glia and neurons
in the cerebrum lacked immunoreactivity of APO2L. Thus, in addition to the
CD95/CD95L system, a second death ligand/death receptor pair may regulate
susceptibility to apoptosis in human glial neoplasms.

L4 ANSWER 7 OF 11 MEDLINE
AN 1999003284 MEDLINE
DN 99003284
TI Interleukin-1 protects transformed keratinocytes from tumor necrosis
factor- ***related*** apoptosis-inducing ligand.
AU Kothny-Wilkes G; Kulms D; Poppelmann B; Luger T A; Kubin M; Schwarz T
CS Department of Dermatology, Ludwig Boltzmann Institute for Cell Biology and
Immunobiology of the Skin, University of Munster, Von-Esmarchstrasse 56,
D-48149 Munster, Germany.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Oct 30)

273 (44) 29247-53.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199902
EW 19990204
AB Tumor necrosis factor- ***related*** apoptosis-inducing ligand (TRAIL)
is a member of the ***tumor*** ***necrosis*** ***factor***
family . It induces apoptosis primarily of transformed but not of
normal cells and may therefore be a promising anti-cancer drug. Studying
the role of TRAIL in apoptosis of keratinocytes, we detected TRAIL
transcripts and protein in both normal human keratinocytes and transformed
keratinocyte cell lines HaCaT and KB. Although normal keratinocytes were
resistant to TRAIL, HaCaT and KB cells underwent apoptosis following TRAIL
exposure. When HaCaT and KB cells were pretreated with the
pro-inflammatory cytokine interleukin-1 (IL-1), cells became resistant to
TRAIL-induced apoptosis. IL-1 significantly induced activation of the
transcription factor NFkappaB in transformed keratinocytes. Moreover, the
proteasome inhibitor MG132, which inhibits IL-1-induced NFkappaB
activation, completely prevented the protective effect of IL-1. Thus, IL-1
appears to protect transformed keratinocytes from the cytotoxic effect of
TRAIL via activation of NFkappaB. These data suggest that NFkappaB
activation may protect cells from TRAIL-induced apoptosis and indicate a
TRAIL receptor-independent pathway, which allows cells to escape the
cytotoxic effect of TRAIL. Because IL-1 is secreted by a variety of tumor
cells and is also released by inflammatory cells participating in the
tumor-host immune response, tumors under these conditions could become
resistant to TRAIL.

L4 ANSWER 8 OF 11 MEDLINE
AN 1998288312 MEDLINE
DN 98288312
TI ERICE, a novel FLICE-activatable caspase.
AU Humke E W; Ni J; Dixit V M
CS Department of Cellular and Molecular Biology, University of Michigan
Medical School, Ann Arbor, Michigan 48109, USA.
NC R01 AG13671 (NIA)
5T32 GM07863-16 (NIGMS)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Jun 19) 273 (25) 15702-7.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-AF078533
EM 199809
AB Programmed cell death, or apoptosis, is a process of fundamental
importance to cellular homeostasis in metazoan organisms (Ellis, R. E.,
Yuan, J., and Horvitz, H. R. (1991) Annu. Rev. Cell Biol. 7, 663-698). The
caspase family of mammalian proteases, ***related*** to the nematode
death protein CED-3, plays a crucial role in apoptosis and inflammation.
We report here the isolation and characterization of a new caspase,
tentatively termed ERICE (Evolutionarily ***Related***
Interleukin-1beta Converting Enzyme). Based on phylogenetic analysis,
ERICE (caspase-13) is a member of the ICE subfamily of caspases which
includes caspase-1 (ICE), caspase-4 (ICErel-II, TX, ICH-2), and caspase-5
(ICErel-III, TY). Overexpression of ERICE induces apoptosis of 293 human
embryonic kidney cells and MCF7 breast carcinoma cells. Like other members
of the subfamily, ERICE is not activated by the serine protease granzyme
B, a caspase-activating component of cytotoxic T cell granules. Therefore,
ERICE most likely does not play a role in granzyme B-induced cell death.
ERICE, however, was activated by caspase-8 (FLICE, MACH, Mch-5), the
apical caspase activated upon engagement of death receptors belonging to
the ***tumor*** ***necrosis*** ***factor*** ***family*** .
This is consistent with a potential role for ERICE in this
receptor-initiated death pathway.

L4 ANSWER 9 OF 11 MEDLINE
AN 1998269066 MEDLINE
DN 98269066
TI Molecular mechanisms of promoter regulation of the gp34 gene that is
trans-activated by an oncoprotein Tax of human T cell leukemia virus type
I.
AU Ohtani K; Tsujimoto A; Tsukahara T; Numata N; Miura S; Sugamura K;
Nakamura M
CS Human Gene Sciences Center, Japan.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Jun 5) 273 (23) 14119-29.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-AB007839
EM 199809
EW 19980903
AB We investigated the molecular mechanism of transcriptional activation of
the gp34 gene by the Tax oncoprotein of human T cell leukemia virus type I
(HTLV-I). gp34 is a type II transmembrane molecule belonging to the
tumor ***necrosis*** ***factor*** ***family*** and is
constitutively expressed on HTLV-I-producing cells but not normal resting
T cells. The transcriptional regulatory region of the gp34 gene was
activated by HTLV-I Tax in the human T cell line Jurkat, in which
endogenous gp34 is induced by Tax. Sequence analysis demonstrated that two
NF-kappaB-like elements (1 and 2) were present in the regulatory region.
Both NF-kappaB-like elements were able to bind to NF-kappaB or its
related factor(s) in a Tax-dependent manner. Chloramphenicol
acetyltransferase assays indicated that NF-kappaB-like element 1 was
Tax-responsive, although the activity was lower than that the native
promoter. NF-kappaB-like element 2 elevated promoter activity when
combined with NF-kappaB-like element 1, indicating cooperative function of
the elements for maximum promoter function. Unlike typical NF-kappaB
elements, the NF-kappaB-like elements in gp34 were not activated by
treatment of Jurkat cells with phorbol ester despite induction of the
NF-kappaB-like binding activity. Chloramphenicol acetyltransferase
reporter assays using the region upstream of the NF-kappaB-like elements
identified an upstream region that reduced transcription from cognate and
noncognate core promoters in a Tax-independent manner. Our results imply
complex regulation of expression of the gp34 gene and suggest implication
of gp34 in proliferation of HTLV-I infected T cells.

L4 ANSWER 10 OF 11 MEDLINE
AN 1998039318 MEDLINE
DN 98039318
TI Apoptotic signaling in lymphocytes.
AU Rudin C M; Van Dongen J; Thompson C B
CS Gwenn Knapp Center for Lupus and Immunology Research, University of
Chicago, IL 60637-5420, USA.
SO CURRENT OPINION IN HEMATOLOGY, (1996 Jan) 3 (1) 35-40. Ref: 28
Journal code: CN0. ISSN: 1065-6251.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199802
EW 19980204
AB Two families of cell surface receptors are integral to the control of
lymphocyte survival and programmed cell death (apoptosis): the tumor
necrosis factor receptor family and the CD28/CTLA4 family. Tumor necrosis
factor receptor family members bind a ***related*** collection of
ligands (the ***tumor*** ***necrosis*** ***factor*** ***family***) that can either induce or inhibit cell death. Two of the
tumor necrosis factor receptor family members, tumor necrosis factor
receptor 1 and Fas, have been implicated in the termination of immune
responses through their ability to induce apoptosis. A number of
cytoplasmic proteins implicated in signal generation by these receptors
recently have been identified. These proteins fall into several
related classes sharing intriguing structural motifs. The CD28 and
CTLA4 molecules share at least two extracellular ligands and signaling
through the two receptors appears to determine the apoptotic sensitivity
of activated T cells. The effects of CD28 and CTLA4 on cell survival are
dependent on T-cell antigen receptor engagement, providing a potent
mechanism for clonally specific T-cell expansion or deletion. The study of
the apoptotic pathways in lymphocytes has led to a better understanding of
the mechanisms of autoimmune disease and serves as a model system for the
study of the regulation of cell survival and tissue homeostasis.

L4 ANSWER 11 OF 11 MEDLINE
AN 97390509 MEDLINE
DN 97390509
TI Control of TRAIL-induced apoptosis by a family of signaling and decoy
receptors [see comments].
CM Comment in: Science 1997 Aug 8;277(5327):768
AU Sheridan J P; Marsters S A; Pitti R M; Gurney A; Skubatch M; Baldwin D;
Ramakrishnan L; Gray C L; Baker K; Wood W I; Goddard A D; Godowski P;
Ashkenazi A
CS Department of Molecular Oncology, Genentech, South San Francisco, CA
94080-4918, USA.
SO SCIENCE, (1997 Aug 8) 277 (5327) 818-21.
Journal code: UJ7. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Cancer Journals; Priority Journals
OS GENBANK-AF012535; GENBANK-AF012536
EM 199710
AB TRAIL (also called Apo2L) belongs to the ***tumor*** ***necrosis***
factor ***family*** , activates rapid apoptosis in tumor cells,
and binds to the death-signaling receptor DR4. Two additional TRAIL
receptors were identified. The receptor designated death receptor 5 (DR5)
contained a cytoplasmic death domain and induced apoptosis much like DR4.
The receptor designated decoy receptor 1 (DcR1) displayed properties of a
glycophospholipid-anchored cell surface protein. DcR1 acted as a decoy
receptor that inhibited TRAIL signaling. Thus, a cell surface mechanism
exists for the regulation of cellular responsiveness to pro-apoptotic
stimuli.

=> file medline embase biosis inpadoc caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	4.98	5.13

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FILE 'INPADOC' ENTERED AT 10:39:33 ON 10 JUL 2000
COPYRIGHT (C) 2000 European Patent Office, Vienna (EPO)

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=> d his

(FILE 'HOME' ENTERED AT 10:33:47 ON 10 JUL 2000)

FILE 'MEDLINE' ENTERED AT 10:33:58 ON 10 JUL 2000
L1 1 S TRELL/AB,BI
L2 0 S TUMOR NECROSIS FACTOR FAMILY RELATED PROTEIN#/AB,BI
L3 53 S TUMOR NECROSIS FACTOR FAMILY/AB,BI
L4 11 S L3 AND RELATED/AB,BI

FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 10:39:33 ON 10
JUL 2000

=> s l1 or l2

'AB' IS NOT A VALID FIELD CODE
4 FILES SEARCHED...
L5 9 L1 OR L2

=> dup rem l5

PROCESSING COMPLETED FOR L5
L6 8 DUP REM L5 (1 DUPLICATE REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 8 ANSWERS - CONTINUE? Y/(N):y

L6 ANSWER 1 OF 8 INPADOC COPYRIGHT 2000 EPO

LEVEL 1
AN 127892689 INPADOC ED 20000523 EW 200020 UP 20000523 UW 200020
TI LIGANDO RELACIONADO A FATOR DE NECROSE DE TUMOR.
IN YVES CHICHEPORTICHE; JEFFREY L. BROWNING
INS CHICHEPORTICHE YVES; BROWNING JEFFREY L
PA BIOGEN, INC.; BIOGEN, INC.; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF
GENEVA; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA
PAS BIOGEN INC; FACULTY OF MEDICINE OF THE UNI
PAA US; CH
DT Patent

PIT BRA UNEXAMINED PATENT APPLICATION
PI BR 9711046 A 20000111
AI BR 1997-11046 A 19970807
PRAI US 1996-23541 P 19960807
US 1996-28515 P 19961018
US 1997-40820 P 19970318
WO 1997-US13945 W 19970807
AB Patente de Invenc o: <B>"LIGANDO RELACIONADO A FATOR E NECROSE DE
TUMOR"<D>. Ligando relacionado a fator de necrose de tumor (***TRELL***), um novo membro da familia de fator de necrose de tumor
(TNF), ***TRELL*** modificado, e composic es farmac uticas compreendendo os mesmos.

L6 ANSWER 2 OF 8 INPADOC COPYRIGHT 2000 EPO

LEVEL 2
AN 44303990 INPADOC EW 199923 UW 199926
TI TUMORNEKROSEFAKTOR-RELATERT LIGAND (***TRELL***), ET NYTT MEDLEM AV
TUMORNEKROSEFAKTORFAMILIEN (TNF), MODIFISERT ***TRELL*** OG FARMAS
YTISKE SAMMENSETNINGER INNEHOLDENDE SLIKE
IN CHICHEPORTICHE, YVES; BROWNING, JEFFREY L.
INS CHICHEPORTICHE YVES; BROWNING JEFFREY L
INA CH; US
PA BIOGEN INC
PAS BIOGEN INC
PAA US
DT Patent
PIT NOA0 APPLICATION FILED
PI NO 9900550 A0 19990205
AI NO 1999-550 A 19990205
PRAI US 1996-23541 P 19960807
US 1996-28515 P 19961018
US 1997-40820 P 19970318
WO 1997-US13945 W 19970807

L6 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1998:112459 CAPLUS
DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a tumor
necrosis factor-related ligand or its receptor
IN Chicheportiche, Yves; Browning, Jeffrey L.
PA Biogen, Inc., USA; Faculty of Medicine of the University of Geneva;
Chicheportiche, Yves; Browning, Jeffrey L.
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE
---------- --- ------ ---------- ------
PI WO 9805783 A1 19980212 WO 1997-US13945 19970807
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ,
LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US,
UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR,
GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA,
GN, ML, MR, NE, SN, TD, TG
AU 9738294 A1 19980225 AU 1997-38294 19970807
CN 1232503 A 19991020 CN 1997-198401 19970807
EP 956351 A1 19991117 EP 1997-935334 19970807
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO
BR 9711046 A 20000111 BR 1997-11046 19970807
NO 9900550 A 19990406 NO 1999-550 19990205
PRAI US 1996-PV23541 19960807
US 1996-PV28515 19961018
US 1997-PV40820 19970318
WO 1997-US13945 19970807
AB Tumor necrosis factor-related ligand (***TRELL***), a novel member of
the tumor necrosis factor family (TNF), modified ***TRELL*** , and
pharmaceutical compns. comprising them. The ***TRELL*** protein or
its receptor may have anti-cancer and/or immunoregulatory applications.
Human cells transfected with the ***TRELL*** gene may be used in gene
therapy to treat tumors, autoimmune and inflammatory disease or inherited
genetic disorders. ***TRELL*** -specific monoclonal antibodies and
antisense RNA against ***TRELL*** are also claimed. The methodol. is
exemplified by treating human adenocarcinoma cells with ***TRELL*** or
TRELL homologs.

L6 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:497233 BIOSIS
DN PREV199799796436
TI Morphotaxonomical studies of Furcraea (Agavaceae) of India.
AU Khan, Hafiz Ahmed
CS Birbal Sahni Inst. Palaebotany, 53 University Rd., Lucknow 226007 India
SO Journal of Plant Anatomy and Morphology (Jodhpur), (1997) Vol. 7, No. 2,
pp. 140-147.
ISSN: 0256-436X.
DT Article; (TAXONOMIC KEY)
LA English
AB Few species of Furcraea Vent. have been introduced in India as garden and
hedge plants, and for obtaining fibre. These are succulent plants like
Agave and are growing in dry, tropical and subtropical places throughout
the country. F. gigantea Vent. is a common species and a more important
plant known as Mauritius Hemp. Other species grown in India are F.
bedinghausii Koch., F. longaeva Karw. & Zucc. F. selloa C. Koch. var.
marginata ***Trell*** . and F. hexapetala Urb. The botanical identity
of south Indian species known as Mauritius Hemp is F. hexapetala Urb.
(Syn. F. cubensis Haw.) and not F. gigantea Vent. The F. gigantea is a
large shrub with fleshy leaves possessing a brown tip spine and armed or
often Basal part only, armed margins. Trunk is long below the rosette of
leaves. A variety of F. gigantea is mediopecta which is variegated with
butter coloured straps along the leaves. This variety is generally grown
as ornamental in the gardens in pots or on the ground. Leaves of
willemetiana, the other variety are light green coloured, armed with
prickles and the juice is of mild odour. Variety marginata of F. selloa
has the leaf margins armed with distant brown horny hooked prickles.

L6 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1991:141403 CAPLUS
DN 114:141403
TI Meningococcal class 1 outer-membrane protein vaccine
IN Seid, Robert C., Jr.; Paradiso, Peter R.; Poolman, Jan T.; Hoogerhout,
Peter; Wiertz, Emmanuel J. H. J.; Van der Ley, Peter; Heckels, John
Edward; Clarke, Ian Nicholas
PA Praxis Biologics, Inc., USA; Rijksinstituut voor Volksgezondheid en
Milieuhygiene
SO PCT Int. Appl., 121 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE
---------- --- ------ ---------- ------

PI WO 9006696 A2 19900628 WO 1989-US5678 19891219
WO 9006696 A3 19900712
W: AU, DK, FI, JP, NO, US
RW: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE
NL 8803111 A 19900716 NL 1988-3111 19881219
NL 8900036 A 19900716 NL 1989-36 19890106
NL 8901612 A 19900716 NL 1989-1612 19890626
AU 9048219 A1 19900710 AU 1990-48219 19891219
AU 640118 B2 19930819
EP 449958 A1 19911009 EP 1990-901397 19891219
EP 449958 B1 19950322
R: AT, BE, CH, DE, ES, FR, GB, IT, LI, LU, NL, SE
JP 06503465 T2 19940421 JP 1990-501662 19891219
AT 120093 E 19950415 AT 1990-901397 19891219
ES 2070312 T3 19950601 ES 1990-901397 19891219
CA 2007248 AA 19900706 CA 1990-2007248 19900105
NO 9102369 A 19910806 NO 1991-2369 19910618
DK 9101174 A 19910815 DK 1991-1174 19910618
PRAI NL 1988-3111 19881219
NL 1989-30 19890106
NL 1989-1612 19890626
NL 1989-36 19890106
WO 1989-US5678 19891219
AB Outer-membrane vesicles, class 1 outer-membrane proteins (OMPs) of
Neisseria meningiditis, fragments or oligopeptide contg. epitopes of the
class 1 OMPs, and antigenic conjugates are provided for immunization
against meningococcal disease. Also provided are cloning and prodn. of
fusion proteins contg. class 1 OMP epitopes and flagellin protein.
Epitope sequences are identified, and DNA sequencing of class 1 OMP genes
from different N. Meningitidis serosubtypes is presented. Thus,
recombinant flagellins contg. either a VR1 (1st variable region of class 1
OMP), VR2, or a cassette of both VR1 and VR2 are effective in eliciting
antibody response which was cross-reactive to purified P1.16 (class 1 OMP
subtype) and, to a lesser extent, to outer-membrane complex. Each
construction also induced significant anti-flagellin titers; control wild
type flagellin only induced antibody response to flagellin itself.
Recombinant flagellin-oligosaccharide conjugate also prepd. and tested.

L6 ANSWER 6 OF 8 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 85035271 EMBASE
DN 1985035271
TI The hypertensive genotype.
AU Harvald B.
CS Odense University Hospital, Dept. Intern. Med. C, DK-5000 Odense, Denmark
SO Scandinavian Journal of Primary Health Care, (1984) 2/3 (96-97). CODEN: SJPCD7
CY Sweden
DT Journal
FS 022 Human Genetics
017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
006 Internal Medicine
LA English
AB ***Trell*** and collaborators have tried to define the hypertensive
genotype by an analysis of risk factors in hypertensive patients with a
varying degree of genetic predisposition. The data support the view that
the genetic predisposition for hypertension is not per se associated with
such accepted cardiovascular risk factors in the population as high serum
cholesterol and triglyceride content, and impaired glucose tolerance. What
is this polygenic predisposition to hypertension like? Gradually it has
proved possible to define some contributing factors. Increased sensitivity
to sodium loading - the mechanism known to be active in some strains of
rats - may result in hypertension in humans as well. An elevated
intracellular sodium concentration with increased smooth muscle
reactiveness has been demonstrated in hypertensive patients. Data are in
existence supporting a correlation between hypertension and a number of
varying traits: Certain HLA-alleles, the C3F-allele in the complement
system, different autoantibodies, herpesvirus antibodies, increased
adrenal responsiveness to angiotensin-II, increased catecholamine release
during exercise, a high proportion of fast twitch fibres in skeletal
muscles. Probably this spectrum of characteristics will be further
broadened in the future. The genetic predisposition to hypertension must
be considered the result of the presence or absence of these traits. The
person who, at the same time, is salt sensitive, C3F positive, with a high
proportion of fast twitch muscle fibres, etc is particularly predisposed.
Today it is not possible to single out the relative importance of
individual factors in the pathogenesis of human hypertension. Nor can we
predict to what extent a diagnostic disentanglement along these lines
should determine the therapeutic strategy.

L6 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1982:174918 BIOSIS
DN BA73:34902
TI HIRSUTINOLIDES FROM VERNONIA-SPP.
AU BOHLMANN F; MUELLER L; GUPTA R K; KING R M; ROBINSON H
CS INST. ORG. CHEM., TECHNICAL UNIV. BERLIN, D-1000 BERLIN 12, W. GER.
SO PHYTOCHEMISTRY (OXF), (1981) 20 (9), 2233-2238. CODEN: PYTCAS. ISSN: 0031-9422.
FS BA; OLD
LA English
AB Of the 19 spp. of Vernonia [V. alemeda H. Robins., V. condensata Baker, V.
corizcea Less, V. echitifolia Mart., V. farinosa Baker, V. gigantea ***Trell*** . Branner et Cor., V. hagei H. Robins., V. holosericea Mart.
ex DC, V. intermedia DC, V. kuntzei Hieron., V. laxa Gardn., V. mariana
Mart., V. missionis Gardn., V. myrsinitis Ekman, V. obtusata Less, V.
regis H. Robins., V. teixeirae H. Robins., V. tomentella Mart. and V.
venosissima Sch. Bip. ex Baker] studied, 5 contained highly oxygenated
sesquiterpene lactones; the rest contained predominantly triterpenes,
especially lupane derivatives.

L6 ANSWER 8 OF 8 MEDLINE DUPLICATE 1
AN 76058643 MEDLINE
DN 76058643
TI Hydantoin derivatives and malignancies of the haemopoietic system.
AU Bichel J
SO ACTA MEDICA SCANDINAVICA, (1975 Oct) 198 (4) 327-8. Journal code: 14G. ISSN: 0001-6101.
CY Sweden
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197603
AB Two patients are described who developed malignant lymphoma (lymphosarcoma) after diphenylydantoin therapy because of epilepsy.
Malignant lymphoma in a few patients receiving this medication has been
described earlier. The literature has been reviewed and discussed recently
by Rausing and ***Trell*** (2).

=> e browning j/au

E1 1 BROWNING ILEY B/AU
E2 6 BROWNING IVAN/AU
E3 203 --> BROWNING J/AU
E4 59 BROWNING J A/AU
E5 2 BROWNING J ARTIE/AU

E6 2 BROWNING J B/AU
E7 5 BROWNING J C/AU
E8 103 BROWNING J D/AU
E9 38 BROWNING J E/AU
E10 3 BROWNING J F/AU
E11 28 BROWNING J G/AU
E12 3 BROWNING J H D/AU

=> s e3-e5

L7 264 ("BROWNING J"/AU OR "BROWNING J A"/AU OR "BROWNING J ARTIE"/AU)

=> e browning jeffrey/au

E1 1 BROWNING JEFFERY/AU
E2 1 BROWNING JEFFERY L/AU
E3 37 --> BROWNING JEFFREY/AU
E4 2 BROWNING JEFFREY C/AU
E5 1 BROWNING JEFFREY CHARLES/AU
E6 1 BROWNING JEFFREY D/AU
E7 1 BROWNING JEFFREY J/AU
E8 146 BROWNING JEFFREY L/AU
E9 2 BROWNING JEFFREY LEE/AU
E10 1 BROWNING JENIFER L/AU
E11 1 BROWNING JENNIFER L/AU
E12 1 BROWNING JENNIFER S/AU

=> s e1-e9

L8 192 ("BROWNING JEFFERY"/AU OR "BROWNING JEFFERY L"/AU OR "BROWNING JEFFREY"/AU OR "BROWNING JEFFREY C"/AU OR "BROWNING JEFFREY CHARLES"/AU OR "BROWNING JEFFREY D"/AU OR "BROWNING JEFFREY J"/AU OR "BROWNING JEFFREY L"/AU OR "BROWNING JEFFREY LEE"/AU)

=> s l7 or l8

L9 456 L7 OR L8

=> s l9 and tumor necrosis factor/ab,bi

'AB' IS NOT A VALID FIELD CODE
L10 74 L9 AND TUMOR NECROSIS FACTOR/AB,BI

=> s l10 and family/ab,bi

'AB' IS NOT A VALID FIELD CODE
L11 26 L10 AND FAMILY/AB,BI

=> dup rem l11

PROCESSING COMPLETED FOR L11
L12 16 DUP REM L11 (10 DUPLICATES REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 16 ANSWERS - CONTINUE? Y/(N):y

L12 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:329685 CAPLUS
DN 131:115182
TI Signaling through the lymphotoxin-.beta. receptor stimulates HIV-1
replication alone and in cooperation with soluble or membrane-bound
TNF-.alpha.
AU Marshall, William L.; Brinkman, Brigitta M. N.; Ambrose, Christine M.;
Pesavento, Patricia A.; Uglialoro, Adele M.; Teng, Edna; Finberg, Robert
W.; ***Browning, Jeffrey L.*** ; Goldfeld, Anne E.
CS Division of Adult Oncology, Dana-Farber Cancer Institute, Boston, MA,
02115, USA
SO J. Immunol. (1999), 162(10), 6016-6023
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB The level of ongoing HIV-1 replication within an individual is crit. to
HIV-1 pathogenesis. Among host immune factors, the cytokine TNF-.alpha.
has previously been shown to increase HIV-1 replication in various
monocyte and T cell model systems. Here, the authors demonstrate that
signaling through the TNF receptor ***family*** member, the
lymphotoxin-.beta. (LT-.beta.) receptor (LT-.beta.R), also regulates HIV-1
replication. Furthermore, HIV-1 replication is cooperatively stimulated
when the distinct LT-.beta.R and TNF receptor systems are simultaneously
engaged by their specific ligands. Moreover, in a physiol. coculture
cellular assay system, the authors show that membrane-bound TNF-.alpha.
and LT-.alpha.1.beta.2 act virtually identically to their sol. forms in
the regulation of HIV-1 replication. Thus, co-signaling via the LT-.beta.
and TNF-.alpha. receptors is probably involved in the modulation of HIV-1
replication and the subsequent detn. of HIV-1 viral burden in monocytes.
Intriguingly, surface expression of LT-.alpha.1.beta.2 is up-regulated on
a T cell line acutely infected with HIV-1, suggesting a pos. feedback loop
between HIV-1 infection, LT-.alpha.1.beta.2 expression, and HIV-1
replication. Given the crit. role that LT-.alpha.1.beta.2 plays in
lymphoid architecture, the authors speculate that LT-.alpha.1.beta.2 may
be involved in HIV-assocd. abnormalities of the lymphoid organs.
RE.CNT 65
RE
(1) Amadori, A; Immunol Today 1990, P374 CAPLUS
(2) Balter, M; Science 1996, V274, P1464 CAPLUS
(3) Bazzoni, F; J Inflamm 1995, V45, P221 CAPLUS
(4) Bergelson, J; Science 1992, V255, P1718 CAPLUS
(5) Boussiotis, V; Proc Natl Acad Sci USA 1994, V91, P7007 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
DUPLICATE 1
AN 1999:325998 BIOSIS
DN PREV199900325998
TI BAFF, a novel ligand of the ***tumor*** ***necrosis*** ***factor*** ***family*** , stimulates B cell growth.
AU Schneider, Pascal; MacKay, Fabienne; Steiner, Veronique; Hofmann, Kay;
Bodmer, Jean-Luc; Holler, Nils; Ambrose, Christine; Lawton, Pornsri;
Bixler, Sarah; Acha-Orbea, Hans; Valmori, Danila; Romero, Pedro;
Werner-Favre, Christiane; Zubler, Rudolph H.; ***Browning, Jeffrey L.***
; Tschopp, Jurg (1)
CS (1) Institute of Biochemistry, University of Lausanne, Ch. des Boveresses
155, CH-1066, Epalinges Switzerland
SO Journal of Experimental Medicine, (June 11, 1999) Vol. 189, No. 11, pp.
1747-1756.
ISSN: 0022-1007.
DT Article
LA English
SL English
AB Members of the ***tumor*** ***necrosis*** ***factor*** (TNF)
family induce pleiotropic biological responses, including cell
growth, differentiation, and even death. Here we describe a novel member
of the TNF ***family*** , designated BAFF (for B cell activating factor
belonging to the TNF ***family***), which is expressed by T cells and
dendritic cells. Human BAFF was mapped to chromosome 13q32-34.
Membrane-bound BAFF was processed and secreted through the action of a
protease whose specificity matches that of the furin ***family*** of
proprotein convertases. The expression of BAFF receptor appeared to be
restricted to B cells. Both membrane-bound and soluble BAFF

induced
proliferation of anti-immunoglobulin M-stimulated peripheral blood B
lymphocytes. Moreover, increased amounts of immunoglobulins were found in
supernatants of germinal center-like B cells costimulated with BAFF. These
results suggest that BAFF plays an important role as costimulator of B
cell proliferation and function.

L12 ANSWER 3 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 2
AN 2000:50715 BIOSIS
DN PREV200000050715
TI Mice transgenic for BAFF develop lymphocytic disorders along with
autoimmune manifestations.
AU Mackay, Fabienne (1); Woodcock, Stephen A.; Lawton, Pornsri; Ambrose,
Christine; Baetscher, Manfred; Schneider, Pascal; Tschopp, Jurg;
Browning, Jeffrey L.
CS (1) Biogen, 12 Cambridge Center, Cambridge, MA USA
SO Journal of Experimental Medicine, (Dec. 6, 1999) Vol. 190, No. 11, pp.
1697-1710.
ISSN: 0022-1007.
DT Article
LA English
SL English
AB The cause of many autoimmune and inflammatory diseases is unresolved,
although dysregulated production of ***tumor*** ***necrosis***
factor (TNF) ***family*** members appears to be important in
many cases. BAFF, a new member of the TNF ***family*** , binds to B
cells and costimulates their growth in vitro. Mice transgenic for BAFF
have vastly increased numbers of mature B and effector T cells, and
develop autoimmune-like manifestations such as the presence of high levels
of rheumatoid factors, circulating immune complexes, anti-DNA
autoantibodies, and immunoglobulin deposition in the kidneys. This
phenotype is reminiscent of certain human autoimmune disorders and
suggests that dysregulation of BAFF expression may be a critical element
in the chain of events leading to autoimmunity.

L12 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1998:112459 CAPLUS
DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a
tumor ***necrosis*** ***factor*** -related ligand or its
receptor
IN Chicheportiche, Yves; ***Browning, Jeffrey L.***
PA Biogen, Inc., USA; Faculty of Medicine of the University of Geneva;
Chicheportiche, Yves; Browning, Jeffrey L.
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9805783	A1	19980212	WO 1997-US13945	19970807
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9738294	A1	19980225	AU 1997-38294	19970807
CN 1232503	A	19991020	CN 1997-198401	19970807
EP 956351	A1	19991117	EP 1997-935334	19970807
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 9711046	A	20000111	BR 1997-11046	19970807
NO 9900550	A	19990406	NO 1999-550	19990205
PRAI US 1996-PV23541		19960807		
US 1996-PV28515		19961018		
US 1997-PV40820		19970318		
WO 1997-US13945		19970807		

AB ***Tumor*** ***necrosis*** ***factor*** -related ligand
(TRELL), a novel member of the ***tumor*** ***necrosis***
factor ***family*** (TNF), modified TRELL, and pharmaceutical
compns. comprising them. The TRELL protein or its receptor may have
anti-cancer and/or immunoregulatory applications. Human cells transfected
with the TRELL gene may be used in gene therapy to treat tumors,
autoimmune and inflammatory disease or inherited genetic disorders.
TRELL-specific monoclonal antibodies and antisense RNA against TRELL are
also claimed. The methodol. is exemplified by treating human
adenocarcinoma cells with TRELL or TRELL homologs.

L12 ANSWER 5 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 3
AN 1999:37693 BIOSIS
DN PREV199900037693
TI Both the lymphotoxin and ***tumor*** ***necrosis*** ***factor***
pathways are involved in experimental murine models of colitis.
AU Mackay, Fabienne (1); ***Browning, Jeffrey L.*** ; Lawton, Pornsri;
Shah, Samir A.; Comiskey, Martina; Bhan, Atul K.; Mizoguchi, Emiko;
Terhorst, Cox; Simpson, Stephen J.
CS (1) Biogen, 12 Cambridge Center, Cambridge, MA 02142 USA
SO Gastroenterology, (Dec., 1998) Vol. 115, No. 6, pp. 1464-1475.
ISSN: 0016-5085.
DT Article
LA English
AB Background & Aims: Membrane lymphotoxin (LT) alpha/beta, a member of the
tumor ***necrosis*** ***factor*** (TNF) ***family***
of immune regulatory molecules, is involved both in the development of
secondary lymphoid tissues and the maintenance of organized lymphoid
tissues in the adult. Defects observed in the mucosal immune system in
animals with a genetically disrupted LTalpha/beta pathway coupled with the
expression of LTalpha/beta in activated T cells motivated an examination
of the importance of this pathway in experimental colitis. Methods:
Soluble LTbeta receptor (LTbetaR) immunoglobulin fusion protein was used
to inhibit the LTalpha/beta light axis in two independent rodent models of
colitis: CD45RBhi CD4+-reconstituted SCID mice and bone marrow
transplanted tgepsilon26 mice (BM fwdarw tgepsilon26). Results: Treatment
with LTbetaR immunoglobulin attenuated the development of both the
clinical and histological manifestations of the disease in these two
murine models of colitis. Given the success of TNF inhibitors in the
treatment of human Crohn's disease, the effects of LTbetaR immunoglobulin
have been compared with antibody to TNF in the BM fwdarw tgepsilon26
model, and both treatments were equally efficacious. Conclusions: The LT
pathway plays a role in the development of colitis as important as that of
the TNF system and, therefore, represents a potential novel

intervention
point for the treatment of inflammatory bowel disease.

L12 ANSWER 6 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:458071 BIOSIS
DN PREV199800458071
TI Caspase-dependent and -independent apoptosis induced by signaling through
TNF ***family*** receptors.
AU ***Browning, Jeffrey L.*** ; Wilson, Cheryl A.
CS Dep. Cell Biol. Immunol. Inflammation, Biogen, Cambridge, MA 02142 USA
SO Journal of Interferon and Cytokine Research, (May, 1998) Vol. 18, No. 5,
pp. A64.
Meeting Info.: 7th International Conference on Tumor Necrosis Factor and
Related Molecules Scientific Advances and Medical Applications Hyannis,
Massachusetts, USA May 17-21, 1998
ISSN: 1079-9907.
DT Conference
LA English

L12 ANSWER 7 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 4
AN 1998:71312 BIOSIS
DN PREV199800071312
TI TWEAK, a new secreted ligand in the ***tumor*** ***necrosis***
factor ***family*** that weakly induces apoptosis.
AU Chicheportiche, Yves; Bourdon, Paul R.; Xu, Haoda; Hsu, Yen-Ming; Scott,
Hamish; Hession, Catherine; Garcia, Irene; ***Browning, Jeffrey L.***
*** (1)***
CS (1) Biogen, 12 Cambridge Cent., Cambridge, MA 02142 USA
SO Journal of Biological Chemistry, (Dec. 19, 1997) Vol. 272, No. 51, pp.
32401-32410.
ISSN: 0021-9258.
DT Article
LA English
AB The members of the ***tumor*** ***necrosis*** ***factor***
(TNF) ***family*** play pivotal roles in the regulation of the immune
system. Here we describe a new ligand in this ***family*** , designated
TWEAK. The mouse and human versions of this protein are unusually
conserved with 93% amino acid identity in the receptor binding domain. The
protein was efficiently secreted from cells indicating that, like TNF,
TWEAK may have the long range effects of a secreted cytokine. TWEAK
transcripts were abundant and found in many tissues, suggesting that TWEAK
and TRAIL belong to a new group of widely expressed ligands. Like many
members of the TNF ***family*** , TWEAK was able to induce
interleukin-8 synthesis in a number of cell lines. The human
adenocarcinoma cell line, HT29, underwent apoptosis in the presence of
both TWEAK and interferon-gamma. Thus, TWEAK resembles many other TNF
ligands in the capacity to induce cell death; however, the fact that
TWEAK-sensitive cells are relatively rare suggests that TWEAK along with
lymphotoxins alpha/beta and possibly CD30L trigger death via a weaker,
non-death domain-dependent mechanism.

L12 ANSWER 8 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 5
AN 1997:177332 BIOSIS
DN PREV199799469045
TI TRAMP, a novel apoptosis-mediating receptor with sequence homology to
tumor ***necrosis*** ***factor*** receptor 1 and Fas(Apo-1/CD95.
AU Bodmer, Jean-Luc (1); Burns, Kim (1); Schneider, Pascal (1); Hofmann, Kay;
Steiner, Veronique (1); Thome, Margot (1); Bornand, Thierry; Hahne,
Michael; Schroeter, Michael; Becker, Karin; Wilson, Anne; French, Lars E.;
Browning, Jeffrey L. ; MacDonald, H. Robson; Tschopp, Jurg
CS (1) Inst. Biochem., Lausanne Branch, Univ. Lausanne, Lausanne Switzerland
SO Immunity, (1997) Vol. 6, No. 1, pp. 79-88.
ISSN: 1074-7613.
DT Article
LA English
AB A novel member of the ***tumor*** ***necrosis*** ***factor***
(TNF) receptor ***family*** , designated TRAMP, has been identified.
The structural organization of the 393 amino acid long human TRAMP is most
homologous to TNF receptor 1. TRAMP is abundantly expressed on thymocytes
and lymphocytes. Its extracellular domain is composed of four
cysteine-rich domains, and the cytoplasmic region contains a death domain
known to signal apoptosis. Overexpression of TRAMP leads to two major
responses, NF-kappa-B activation and apoptosis. TRAMP-induced cell death
is inhibited by an inhibitor of ICE-like proteases, but not by Bcl-2. In
addition, TRAMP does not appear to interact with any of the known
apoptosis-inducing ligands of the TNF ***family*** .

L12 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1997:127471 CAPLUS
DN 126:135644
TI Complexes of modified lymphotoxins as pharmaceutical preparations
IN ***Browning, Jeffrey L.*** ; Meier, Werner; Karpusas, Michael N.
PA Biogen, Inc., USA
SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9640774	A1	19961219	WO 1996-US9773	19960606

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE,
ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS,
LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD,
SE, SG
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR,
IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN
AU 9661663 A1 19961230 AU 1996-61663 19960606
PRAI US 1995-476074 19950607
WO 1996-US9773 19960606
AB This invention related to lymphotoxin (LT) complexes comprising
lymphotoxin-.alpha. (LT-.alpha.) and lymphotoxin-.beta. (LT-.beta.)
subunits, and modified versions thereof, which can act as specific
inhibitors of the biol. events mediated by the ligands and receptors of
the ***tumor*** ***necrosis*** ***factor*** (TNF)
family . This invention also relates to unique portions of the
LT-.alpha. and LT-.beta. protein sequences =, "LT subunit assocn.
domains", which potentiate subunit assembly into an active trimeric
ligand. This invention provides TNF-related ligand monomers mutated in
their resp. subunit assocn. domains which permits them to form heteromeric
complexes with LT subunits. Altered ligands which have only one
functional receptor binding site per heteromer can inhibit signaling by
that receptor. Also provided are mutant and chimeric LT subunits with can

alter the receptor binding properties of heteromeric complexes assembled
from the. Polypeptides comprising LT subunit assocn. domains, LT
heteromeric complexes which inhibit receptor signaling, pharmaceutical
compns. comprising LT heteromeric inhibitors, and methods for treatment
using those pharmaceutical compns. are also provided.

L12 ANSWER 10 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 6
AN 1996:521458 BIOSIS
DN PREV199699243814
TI Lymphotoxin beta receptor triggering induces activation of the nuclear
factor kappa-B transcription factor in some cell types.
AU Mackay, Fabienne (1); Majeau, Gerard R.; Hochman, Paula S.; ***Browning,***
*** Jeffrey L.***
CS (1) Dep. Cell Biol., Biogen Inc., 12 Cambridge Cent., Cambridge, MA 02142
USA
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 40, pp. 24934-24938.
ISSN: 0021-9258.
DT Article
LA English
AB NF-kappa-B is a pleiotropic transcription factor capable of activating the
expression of a great variety of genes critical for the immunoinflammatory
response. ***Tumor*** ***necrosis*** ***factor*** alpha
(TNF-alpha) and lymphotoxin alpha (LT-alpha, originally TNF-beta) are
potent nuclear factor kappa-B (NF-kappa-B) activators in various cell
types. The LT-alpha molecule, in addition to being secreted as a soluble
trimer, can also form membrane-anchored heterotrimers with the LT-beta
chain, another member of the TNF ***family*** . The LT-alpha-1-beta-2
heterotrimer binds a specific receptor, called the LT-beta receptor
(LT-beta-R), which is also a member of the TNF receptor ***family*** .
Here, we show that engagement of LT-beta-R with a soluble form of
LT-alpha-1-beta-2 or with a specific anti-LT-beta-R agonistic monoclonal
antibody CBE11 quickly induces activation of NF-kappa-B in HT-29 and WiDr
human adenocarcinomas. LT-beta-R triggering activates NF-kappa-B and
induces proliferation in WI-38 human lung fibroblasts. No NF-kappa-B
activation is observed in human umbilical vein endothelial cells,
correlating with the inability of LT-beta-R activation to induce
expression of NF-kappa-B-dependent cell surface adhesion molecules. Thus,
like several other members of the TNF receptor ***family*** , the
LT-beta-R can activate NF-kappa-B following receptor ligation in some but
not all LT-beta-R-positive cells.

L12 ANSWER 11 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 7
AN 1996:241639 BIOSIS
DN PREV199698789768
TI Preparation and characterization of soluble recombinant heterotrimeric
complexes of human lymphotoxins alpha and beta.
AU ***Browning, Jeffrey L. (1)*** ; Miatkowski, Konrad; Griffiths, David
A.; Bourdon, Paul R.; Hession, Catherine; Ambrose, Christine M.; Meier,
Werner
CS (1) Biogen, 14 Cambridge Cent., Cambridge, MA 02142 USA
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 15, pp. 8618-8626.
ISSN: 0021-9258.
DT Article
LA English
AB The lymphotoxin (LT) protein complex is a heteromer of alpha (LT-alpha,
also called ***tumor*** ***necrosis*** ***factor*** (TNF)beta)
and beta (LT-beta) chains anchored to the membrane surface by the
transmembrane domain of the LT-beta portion. Both proteins belong to the
TNF ***family*** of ligands and receptors that regulate aspects of the
immune and inflammatory systems. The LT complex is found on activated
lymphocytes and binds to the lymphotoxin-beta receptor, which is generally
present on nonlymphoid cells. The signaling function of this
receptor-ligand pair is not precisely known but is believed to be involved
in the development of the peripheral lymphoid organs. To analyze the
properties of this complex, a soluble, biologically active form of the
surface complex was desired. The LT-beta molecule was engineered into a
secreted form and co-expressed with LT-alpha using baculovirus/insect cell
technology. By exploiting receptor affinity columns, the LT-alpha-3,
LT-alpha-2/beta-1, and LT-alpha-1/beta-2 forms were purified. All three
molecules were trimers, and their biochemical properties are described.
The level of LT-alpha-3-like components in the LT-alpha-1/beta-2
preparation was found to be 0.02% by following the activity of the
preparation in a WEHI 164 cytotoxicity assay. LT-alpha-3 with an
asparagine 50 mutation (D50N) cannot bind the TNF receptors. Heteromeric
LT complexes were prepared with this mutant LT-alpha form, allowing a
precise delineation of the extent of biological activity mediated by the
TNF receptors. A LT-alpha-3 based cytotoxic activity was used to show that
the LT-alpha-1/beta-2 form cannot readily scramble into a mixture of forms
following various treatments and storage periods. This biochemical
characterization of the LT heteromeric ligands and the demonstration of
their stability provides a solid foundation for both biological studies
and an analysis of the specificity of the LT-beta and TNF receptors for
the various LT forms.

L12 ANSWER 12 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 8
AN 1996:229916 BIOSIS
DN PREV199698794045
TI Signaling through the lymphotoxin beta receptor induces the death of some
adenocarcinoma tumor lines.
AU ***Browning, Jeffrey L. (1)*** ; Miatkowski, Konrad; Sizing, Irene;
Griffiths, David; Zafari, Mohammad; Benjamin, Christopher D.; Meier,
Werner; Mackay, Fabienne
CS (1) Dep. Immunology Inflammation, Biogen, 14 Cambridge Center, Cambridge,
MA 02142 USA
SO Journal of Experimental Medicine, (1996) Vol. 183, No. 3, pp. 867-878.
ISSN: 0022-1007.
DT Article
LA English
AB Surface lymphotoxin (LT) is a heteromeric complex of LT-alpha and LT-beta
chains that binds to the LT-beta receptor (LT-beta-R), a member of the
tumor ***necrosis*** ***factor*** (TNF) ***family***
of receptors. The biological function of this receptor-ligand system is
poorly characterized. Since signaling through other members of this
receptor ***family*** can induce cell death, e.g., the TNF and Fas
receptors, it is important to determine if similar signaling events can be
communicated via the LT-beta-R. A soluble form of the surface complex was
produced by coexpression of LT-alpha and a converted form of

LT-beta
wherein the normally type II LT-beta membrane protein was changed to a
type I secreted form. Recombinant LT-alpha-1/beta-2 was cytotoxic to the
human adenocarcinoma cell lines HT-29, WiDr, MDA-NB-468, and HT-3 when
added with the synergizing agent interferon (IFN) gamma. When immobilized
on a plastic surface, anti-LT- sbd R, monoclonal antibodies (mAbs) induced
the death of these cells, demonstrating direct signaling via the
LT-beta-R. Anti-LT-beta-R mAbs were also identified that inhibited
ligand-induced cell death, whereas others were found to potentiate the
activity of the ligand when added in solution. The human WiDr
adenocarcinoma line forms solid tumors in immunocompromised mice, and
treatment with an anti-LT-beta-R antibody combined with human IFN-gamma
arrested tumor growth. The delineation of a biological signaling event
mediated by the LT-beta-R opens a window for further studies on its
immunological role, and furthermore, activation of the LT-beta-R may have
an application in tumor therapy.

L12 ANSWER 13 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 9
AN 1994:257127 BIOSIS
DN PREV199497270127
TI A lymphotoxin-beta-specific receptor.
AU Crowe, Paul D.; Vanarsdale, Todd L.; Walter, Barbara N.; Ware, Carl F.
(1); Hession, Catherine; Ehrenfels, Barbara; ***Browning, Jeffrey L.***
; Din, Wenie S.; Goodwin, Raymond G.; Smith, Craig A.
CS (1) Div. Biomed. Sci., Univ. Calif., Riverside, CA 92521 USA
SO Science (Washington D C), (1994) Vol. 264, No. 5159, pp. 707-710.
ISSN: 0036-8075.
DT Article
LA English
AB ***Tumor*** ***necrosis*** ***factor*** (TN F) and lymphotoxin-alpha (LT-alpha) are members of a ***family*** of secreted
and cell surface cytokines that participate in the regulation of immune
and inflammatory responses. The cell surface form of LT-alpha is assembled
during biosynthesis as a heteromeric complex with lymphotoxin-beta
(LT-beta), a type II transmembrane protein that is another member of the
TNF ligand ***family***. Secreted LT-alpha is a homotrimer that binds
to distinct TNF receptors of 60 and 80 kilodaltons; however, these
receptors do not recognize the major cell surface LT-alpha-LT-beta
complex. A receptor specific for human LT-beta was identified, which
suggests that cell surface LT may have functions that are distinct from
those of secreted LT-alpha.

L12 ANSWER 14 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 10
AN 1993:273116 BIOSIS
DN PREV199396003341
TI Lymphotoxin beta, a novel member of the TNF ***family*** that forms a
heteromeric complex with lymphotoxin on the cell surface.
AU ***Browning, Jeffery L. (1)*** ; Ngam-Ek, Apinya (1); Lawton, Pornsri
(1); Demarinis, Janice (1); Tizard, Richard (1); Chow, E. Pingchang (1);
Hession, Catherine (1); O'Brine-Greco, Betsy (1); Foley, Susan F. (1);
Ware, Carl F.
CS (1) Biogen Incorporated, 14 Cambridge Cent., Cambridge, Massachusetts
02142 USA
SO Cell, (1993) Vol. 72, No. 6, pp. 847-856.
ISSN: 0092-8674.
DT Article
LA English
AB The lymphokine ***tumor*** ***necrosis*** ***factor*** (TNF)
has a well-defined role as an inducer of inflammatory responses; however,
the function of the structurally related molecule lymphotoxin (LT-alpha)
is unknown. LT-alpha is present on the surface of activated T, B and LAK
cells as a complex with a 33 kd glycoprotein, and cloning of the cDNA
encoding the associated protein, called lymphotoxin beta (LT-beta),
revealed it to be a type II membrane protein with significant homology to
TNF, LT-alpha, and the ligand for the CD40 receptor. The gene for LT-beta
was found next to the TNF-LT locus in the major histocompatibility complex
(MHC); a region of the MHC with possible linkage to autoimmune disease.
These observations raise the possibility that a surface LT-alpha-LT-beta
complex may have a specific role in immune regulation distinct from the
functions ascribed to TNF.

L12 ANSWER 15 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1993:334815 BIOSIS
DN PREV199345029540
TI Lymphotoxin-beta, a new member of the TNF cytokine ***family***.
AU Ware, C. (1); Crowe, P.; Van Arsdale, T.; Hesson, C.; Tizard, R.; Chow,
P.; ***Browning, J.***
CS (1) Univ. Calif., Riverside, CA 92521 USA
SO Journal of Immunology, (1993) Vol. 150, No. 8 PART 2, pp. 294A.
Meeting Info.: Joint Meeting of the American Association of Immunologists
and the Clinical Immunology Society Denver, Colorado, USA May 21-25, 1993
ISSN: 0022-1767.
DT Conference
LA English

L12 ANSWER 16 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1993:286087 BIOSIS
DN PREV199345004212
TI Lymphotoxin-beta: A new member of the TNF ***family*** that forms a
heteromeric complex with lymphotoxin on the cell surface.
AU ***Browning, Jeffrey L. (1)*** ; Tizard, Richard (1); Ngam-Ek, Apinya
(1); Lawton, Pornsri (1); Demarinis, Janice (1); Chow, E. Pingchang (1);
Hession, Catherine (1); Greco, Betsy (1); Foley, Susan (1); Ware, Carl F.
CS (1) Biogen, Cambridge, MA USA
SO Journal of Cellular Biochemistry Supplement, (1993) Vol. 0, No. 17 PART B,
pp. 87.
Meeting Info.: Keystone Symposium on Cytokines and Cytokine Receptors:
From Cloning to the Clinic Keystone, Colorado, USA January 31-February 7,
1993
ISSN: 0733-1959.
DT Conference
LA English

=> e chichportiche yves/au

E1	1	CHICHOWSKI S/AU
E2	1	CHICHPORSTICH C/AU
E3	0	--> CHICHPORTICHE YVES/AU
E4	1	CHICHTMAN S/AU
E5	1	CHICHTON A/AU
E6	3	CHICHU YOSHIHISA/AU
E7	3	CHICHUA A G/AU
E8	11	CHICHUA A I/AU
E9	2	CHICHUA B K/AU
E10	6	CHICHUA D G/AU
E11	2	CHICHUA D T/AU
E12	2	CHICHUA DAVID G/AU

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(FILE 'HOME' ENTERED AT 10:33:47 ON 10 JUL 2000)

FILE 'MEDLINE' ENTERED AT 10:33:58 ON 10 JUL 2000
L1 1 S TRELL/AB,BI
L2 0 S TUMOR NECROSIS FACTOR FAMILY RELATED PROTEIN#/AB,BI
L3 53 S TUMOR NECROSIS FACTOR FAMILY/AB,BI
L4 11 S L3 AND RELATED/AB,BI

FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 10:39:33 ON 10 JUL 2000
L5 9 S L1 OR L2
L6 8 DUP REM L5 (1 DUPLICATE REMOVED)
E BROWNING J/AU
L7 264 S E3-E5
E BROWNING JEFFREY/AU
L8 192 S E1-E9
L9 456 S L7 OR L8
L10 74 S L9 AND TUMOR NECROSIS FACTOR/AB,BI
L11 26 S L10 AND FAMILY/AB,BI
L12 16 DUP REM L11 (10 DUPLICATES REMOVED)
E CHICHPORTICHE YVES/AU

=> s l10 and trell/ab,bi

'AB' IS NOT A VALID FIELD CODE
L13 1 L10 AND TRELL/AB,BI

=> d bib ab

L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2000 ACS
AN 1998:112459 CAPLUS
DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a ***tumor*** ***necrosis*** ***factor*** -related ligand or its receptor
IN Chicheportiche, Yves; ***Browning, Jeffrey L.***
PA Biogen, Inc., USA; Faculty of Medicine of the University of Geneva; Chicheportiche, Yves; Browning, Jeffrey L.
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9805783	A1	19980212	WO 1997-US13945	19970807

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9738294	A1	19980225	AU 1997-38294	19970807
CN 1232503	A	19991020	CN 1997-198401	19970807
EP 956351	A1	19991117	EP 1997-935334	19970807

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

BR 9711046	A	20000111	BR 1997-11046	19970807
NO 9900550	A	19990406	NO 1999-550	19990205

PRAI US 1996-PV23541 19960807
US 1996-PV28515 19961018
US 1997-PV40820 19970318
WO 1997-US13945 19970807
AB ***Tumor*** ***necrosis*** ***factor*** -related ligand (***TRELL***), a novel member of the ***tumor*** ***necrosis*** ***factor*** family (TNF), modified ***TRELL*** , and pharmaceutical compns. comprising them. The ***TRELL*** protein or its receptor may have anti-cancer and/or immunoregulatory applications. Human cells transfected with the ***TRELL*** gene may be used in gene therapy to treat tumors, autoimmune and inflammatory disease or inherited genetic disorders. ***TRELL*** -specific monoclonal antibodies and antisense RNA against ***TRELL*** are also claimed. The methodol. is exemplified by treating human adenocarcinoma cells with ***TRELL*** or ***TRELL*** homologs.

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E1	22	CHICHEPORTICHE V/AU
E2	41	CHICHEPORTICHE Y/AU
E3	29 -->	CHICHEPORTICHE YVES/AU
E4	1	CHICHEPORTICHES R/AU
E5	1	CHICHEPORTIGHE C/AU
E6	1	CHICHEPROTICHE Y/AU
E7	1	CHICHER J F/AU
E8	16	CHICHER PIERRE/AU
E9	1	CHICHERA GUY/AU
E10	1	CHICHERA M F/AU
E11	1	CHICHERA MIKHAIL F/AU
E12	3	CHICHEREAU CLAIRE/AU

=> s e2-e3

L14 70 ("CHICHEPORTICHE Y"/AU OR "CHICHEPORTICHE YVES"/AU)

=> s l14 and trell/ab,bi

'AB' IS NOT A VALID FIELD CODE
L15 3 L14 AND TRELL/AB,BI

=> dup rem l15

PROCESSING COMPLETED FOR L15
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)

=> d l- bib ab

YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L16 ANSWER 1 OF 3 INPADOC COPYRIGHT 2000 EPO

LEVEL 1
AN 127892689 INPADOC ED 20000523 EW 200020 UP 20000523 UW 200020
TI LIGANDO RELACIONADO A FATOR DE NECROSE DE TUMOR.
IN YVES CHICHEPORTICHE; JEFFREY L. BROWNING
INS ***CHICHEPORTICHE YVES*** ; BROWNING JEFFREY L
PA BIOGEN, INC.; BIOGEN, INC.; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA
PAS BIOGEN INC; FACULTY OF MEDICINE OF THE UNI
PAA US; CH
DT Patent
PIT BRA UNEXAMINED PATENT APPLICATION
PI BR 9711046 A 20000111
AI BR 1997-11046 A 19970807
PRAI US 1996-23541 P 19960807
US 1996-28515 P 19961018
US 1997-40820 P 19970318
WO 1997-US13945 W 19970807
AB Patente de Invenc o: <B>"LIGANDO RELACIONADO A FATOR E NECROSE DE TUMOR"<D>. Ligando relacionado a fator de necrose de tumor (***TRELL***), um novo membro da familia de fator de necrose de tumor (TNF), ***TRELL*** modificado, e composic es farmac uticas compreendendo os mesmos.

L16 ANSWER 2 OF 3 INPADOC COPYRIGHT 2000 EPO

LEVEL 2
AN 44303990 INPADOC EW 199923 UW 199926
TI TUMORNEKROSEFAKTOR-RELATERT LIGAND (***TRELL***), ET NYTT MEDLEM AV TUMORNEKROSEFAKTORFAMILIEN (TNF), MODIFISERT ***TRELL*** OG FARMAS YTISKE SAMMENSETNINGER INNEHOLDENDE SLIKE
IN CHICHEPORTICHE, YVES; BROWNING, JEFFREY L.
INS ***CHICHEPORTICHE YVES*** ; BROWNING JEFFREY L
INA CH; US
PA BIOGEN INC
PAS BIOGEN INC
PAA US
DT Patent
PIT NOA0 APPLICATION FILED
PI NO 9900550 A0 19990205
AI NO 1999-550 A 19990205
PRAI US 1996-23541 P 19960807
US 1996-28515 P 19961018
US 1997-40820 P 19970318
WO 1997-US13945 W 19970807

L16 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2000 ACS
AN 1998:112459 CAPLUS
DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a tumor necrosis factor-related ligand or its receptor
IN ***Chicheportiche, Yves*** ; Browning, Jeffrey L.
PA Biogen, Inc., USA; Faculty of Medicine of the University of Geneva; Chicheportiche, Yves; Browning, Jeffrey L.
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9805783	A1	19980212	WO 1997-US13945	19970807

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9738294	A1	19980225	AU 1997-38294	19970807
CN 1232503	A	19991020	CN 1997-198401	19970807
EP 956351	A1	19991117	EP 1997-935334	19970807

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

BR 9711046	A	20000111	BR 1997-11046	19970807
NO 9900550	A	19990406	NO 1999-550	19990205

PRAI US 1996-PV23541 19960807
US 1996-PV28515 19961018
US 1997-PV40820 19970318
WO 1997-US13945 19970807
AB Tumor necrosis factor-related ligand (***TRELL***), a novel member of the tumor necrosis factor family (TNF), modified ***TRELL*** , and pharmaceutical compns. comprising them. The ***TRELL*** protein or its receptor may have anti-cancer and/or immunoregulatory applications. Human cells transfected with the ***TRELL*** gene may be used in gene therapy to treat tumors, autoimmune and inflammatory disease or inherited genetic disorders. ***TRELL*** -specific monoclonal antibodies and antisense RNA against ***TRELL*** are also claimed. The methodol. is exemplified by treating human adenocarcinoma cells with ***TRELL*** or ***TRELL*** homologs.

=> d his

(FILE 'HOME' ENTERED AT 10:33:47 ON 10 JUL 2000)

FILE 'MEDLINE' ENTERED AT 10:33:58 ON 10 JUL 2000
L1 1 S TRELL/AB,BI
L2 0 S TUMOR NECROSIS FACTOR FAMILY RELATED PROTEIN#/AB,BI
L3 53 S TUMOR NECROSIS FACTOR FAMILY/AB,BI
L4 11 S L3 AND RELATED/AB,BI

FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 10:39:33 ON 10 JUL 2000
L5 9 S L1 OR L2
L6 8 DUP REM L5 (1 DUPLICATE REMOVED)
E BROWNING J/AU
L7 264 S E3-E5
E BROWNING JEFFREY/AU
L8 192 S E1-E9
L9 456 S L7 OR L8
L10 74 S L9 AND TUMOR NECROSIS FACTOR/AB,BI
L11 26 S L10 AND FAMILY/AB,BI
L12 16 DUP REM L11 (10 DUPLICATES REMOVED)
E CHICHPORTICHE YVES/AU
L13 1 S L10 AND TRELL/AB,BI
E CHICHEPORTICHE YVES/AU
L14 70 S E2-E3
L15 3 S L14 AND TRELL/AB,BI
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)

=> s l1

'AB' IS NOT A VALID FIELD CODE
L17 9 L1

=> dup rem l17

PROCESSING COMPLETED FOR L17
L18 8 DUP REM L17 (1 DUPLICATE REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 8 ANSWERS - CONTINUE? Y/(N):y

L18 ANSWER 1 OF 8 INPADOC COPYRIGHT 2000 EPO

LEVEL 1
AN 127892689 INPADOC ED 20000523 EW 200020 UP 20000523 UW 200020
TI LIGANDO RELACIONADO A FATOR DE NECROSE DE TUMOR.
IN YVES CHICHEPORTICHE; JEFFREY L. BROWNING
INS CHICHEPORTICHE YVES; BROWNING JEFFREY L
PA BIOGEN, INC.; BIOGEN, INC.; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA
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DT Patent
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L18 ANSWER 2 OF 8 INPADOC COPYRIGHT 2000 EPO

LEVEL 2
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DT Patent
PIT NOA0 APPLICATION FILED
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DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a tumor
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IN Chicheportiche, Yves; Browning, Jeffrey L.
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SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9805783	A1	19980212	WO 1997-US13945	19970807

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9738294	A1	19980225	AU 1997-38294	19970807
CN 1232503	A	19991020	CN 1997-198401	19970807
EP 956351	A1	19991117	EP 1997-935334	19970807

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

BR 9711046	A	20000111	BR 1997-11046	19970807
NO 9900550	A	19990406	NO 1999-550	19990205

PRAI US 1996-PV23541 19960807
US 1996-PV28515 19961018
US 1997-PV40820 19970318
WO 1997-US13945 19970807

AB Tumor necrosis factor-related ligand (***TRELL***), a novel member of the tumor necrosis factor family (TNF), modified ***TRELL*** , and pharmaceutical compns. comprising them. The ***TRELL*** protein or its receptor may have anti-cancer and/or immunoregulatory applications. Human cells transfected with the ***TRELL*** gene may be used in gene therapy to treat tumors, autoimmune and inflammatory disease or inherited genetic disorders. ***TRELL*** -specific monoclonal antibodies and antisense RNA against ***TRELL*** are also claimed. The methodol. is exemplified by treating human adenocarcinoma cells with ***TRELL*** or ***TRELL*** homologs.

L18 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:497233 BIOSIS
DN PREV199799796436
TI Morphotaxonomical studies of Furcraea (Agavaceae) of India.
AU Khan, Hafiz Ahmed
CS Birbal Sahni Inst. Palaebotany, 53 University Rd., Lucknow 226007 India
SO Journal of Plant Anatomy and Morphology (Jodhpur), (1997) Vol. 7, No. 2, pp. 140-147.
ISSN: 0256-436X.
DT Article; (TAXONOMIC KEY)
LA English
AB Few species of Furcraea Vent. have been introduced in India as garden and hedge plants, and for obtaining fibre. These are succulent plants like Agave and are growing in dry, tropical and subtropical places throughout the country. F. gigantea Vent. is a common species and a more important plant known as Mauritius Hemp. Other species grown in India are F. bedinghausii Koch., F. longaeva Karw. & Zucc. F. selloa C. Koch. var. marginata ***Trell*** . and F. hexapetala Urb. The botanical identity of south Indian species known as Mauritius Hemp is F. hexapetala Urb. (Syn. F. cubensis Haw.) and not F. gigantea Vent. The F. gigantea is a large shrub with fleshy leaves possessing a brown tip spine and armed or often Basal part only, armed margins. Trunk is long below the rosette of leaves. A variety of F. gigantea is mediopecta which is variegated with butter coloured straps along the leaves. This variety is generally grown as ornamental in the gardens in pots or on the ground. Leaves of willemetiana, the other variety are light green coloured, armed with prickles and the juice is of mild odour. Variety marginata of F. selloa has the leaf margins armed with distant brown horny hooked prickles.

L18 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1991:141403 CAPLUS
DN 114:141403
TI Meningococcal class 1 outer-membrane protein vaccine
IN Seid, Robert C., Jr.; Paradiso, Peter R.; Poolman, Jan T.; Hoogerhout, Peter; Wiertz, Emmanuel J. H. J.; Van der Ley, Peter; Heckels, John Edward; Clarke, Ian Nicholas
PA Praxis Biologics, Inc., USA; Rijksinstituut voor Volksgezondheid en Milieuhygiene
SO PCT Int. Appl., 121 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9006696	A2	19900628	WO 1989-US5678	19891219
WO 9006696	A3	19900712		

W: AU, DK, FI, JP, NO, US
RW: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE

NL 8803111	A	19900716	NL 1988-3111	19881219
NL 8900036	A	19900716	NL 1989-36	19890106
NL 8901612	A	19900716	NL 1989-1612	19890626
AU 9048219	A1	19900710	AU 1990-48219	19891219
AU 640118	B2	19930819		
EP 449958	A1	19911009	EP 1990-901397	19891219
EP 449958	B1	19950322		

R: AT, BE, CH, DE, ES, FR, GB, IT, LI, LU, NL, SE

JP 06503465	T2	19940421	JP 1990-501662	19891219

AT 120093 E 19950415 AT 1990-901397 19891219
ES 2070312 T3 19950601 ES 1990-901397 19891219
CA 2007248 AA 19900706 CA 1990-2007248 19900105
NO 9102369 A 19910806 NO 1991-2369 19910618
DK 9101174 A 19910815 DK 1991-1174 19910618
PRAI NL 1988-3111 19881219
NL 1989-30 19890106
NL 1989-1612 19890626
NL 1989-36 19890106
WO 1989-US5678 19891219
AB Outer-membrane vesicles, class 1 outer-membrane proteins (OMPs) of
Neisseria meningiditis, fragments or oligopeptide contg. epitopes of the
class 1 OMPs, and antigenic conjugates are provided for immunization
against meningococcal disease. Also provided are cloning and prodn. of
fusion proteins contg. class 1 OMP epitopes and flagellin protein.
Epitope sequences are identified, and DNA sequencing of class 1 OMP genes
from different N. Meningitidis serosubtypes is presented. Thus,
recombinant flagellins contg. either a VR1 (1st variable region of class 1
OMP), VR2, or a cassette of both VR1 and VR2 are effective in eliciting
antibody response which was cross-reactive to purified P1.16 (class 1 OMP
subtype) and, to a lesser extent, to outer-membrane complex. Each
construction also induced significant anti-flagellin titers; control wild
type flagellin only induced antibody response to flagellin itself.
Recombinant flagellin-oligosaccharide conjugate also prepd. and tested.

L18 ANSWER 6 OF 8 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 85035271 EMBASE
DN 1985035271
TI The hypertensive genotype.
AU Harvald B.
CS Odense University Hospital, Dept. Intern. Med. C, DK-5000 Odense, Denmark
SO Scandinavian Journal of Primary Health Care, (1984) 2/3 (96-97).
CODEN: SJPCD7
CY Sweden
DT Journal
FS 022 Human Genetics
017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
006 Internal Medicine
LA English
AB ***Trell*** and collaborators have tried to define the hypertensive
genotype by an analysis of risk factors in hypertensive patients with a
varying degree of genetic predisposition. The data support the view that
the genetic predisposition for hypertension is not per se associated with
such accepted cardiovascular risk factors in the population as high serum
cholesterol and triglyceride content, and impaired glucose tolerance. What
is this polygenic predisposition to hypertension like? Gradually it has
proved possible to define some contributing factors. Increased sensitivity
to sodium loading - the mechanism known to be active in some strains of
rats - may result in hypertension in humans as well. An elevated
intracellular sodium concentration with increased smooth muscle
reactiveness has been demonstrated in hypertensive patients. Data are in
existence supporting a correlation between hypertension and a number of
varying traits: Certain HLA-alleles, the C3F-allele in the complement
system, different autoantibodies, herpesvirus antibodies, increased
adrenal responsiveness to angiotensin-II, increased catecholamine release
during exercise, a high proportion of fast twitch fibres in skeletal
muscles. Probably this spectrum of characteristics will be further
broadened in the future. The genetic predisposition to hypertension must
be considered the result of the presence or absence of these traits. The
person who, at the same time, is salt sensitive, C3F positive, with a high
proportion of fast twitch muscle fibres, etc is particularly predisposed.
Today it is not possible to single out the relative importance of
individual factors in the pathogenesis of human hypertension. Nor can we
predict to what extent a diagnostic disentanglement along these lines
should determine the therapeutic strategy.

L18 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1982:174918 BIOSIS
DN BA73:34902
TI HIRSUTINOLIDES FROM VERNONIA-SPP.
AU BOHLMANN F; MUELLER L; GUPTA R K; KING R M; ROBINSON H
CS INST. ORG. CHEM., TECHNICAL UNIV. BERLIN, D-1000 BERLIN 12, W. GER.
SO PHYTOCHEMISTRY (OXF), (1981) 20 (9), 2233-2238.
CODEN: PYTCAS. ISSN: 0031-9422.
FS BA; OLD
LA English
AB Of the 19 spp. of Vernonia [V. alemeda H. Robins., V. condensata Baker, V.
corizcea Less, V. echitifolia Mart., V. farinosa Baker, V. gigantea
Trell . Branner et Cor., V. hagei H. Robins., V. holosericea Mart.
ex DC, V. intermedia DC, V. kuntzei Hieron., V. laxa Gardn., V. mariana
Mart., V. missionis Gardn., V. myrsinitis Ekman, V. obtusata Less, V.
regis H. Robins., V. teixeirae H. Robins., V. tomentella Mart. and V.
venosissima Sch. Bip. ex Baker] studied, 5 contained highly oxygenated
sesquiterpene lactones; the rest contained predominantly triterpenes,
especially lupane derivatives.

L18 ANSWER 8 OF 8 MEDLINE DUPLICATE 1
AN 76058643 MEDLINE
DN 76058643
TI Hydantoin derivatives and malignancies of the haemopoietic system.
AU Bichel J
SO ACTA MEDICA SCANDINAVICA, (1975 Oct) 198 (4) 327-8.
Journal code: 14G. ISSN: 0001-6101.
CY Sweden
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197603
AB Two patients are described who developed malignant lymphoma (lymphosarcoma) after diphenylydantoin therapy because of epilepsy.
Malignant lymphoma in a few patients receiving this medication has been
described earlier. The literature has been reviewed and discussed recently
by Rausing and ***Trell*** (2).

=> file stnguide

COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 128.86 133.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
SINCE FILE ENTRY TOTAL SESSION
CA SUBSCRIBER PRICE -5.01 -5.01

FILE 'STNGUIDE' ENTERED AT 10:52:27 ON 10 JUL 2000
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COPYRIGHT (C) 2000 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 7, 2000 (20000707/UP).

=> file medline

COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.00 133.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
SINCE FILE TOTAL
ENTRY SESSION
CA SUBSCRIBER PRICE 0.00 -5.01

FILE 'MEDLINE' ENTERED AT 10:56:01 ON 10 JUL 2000

FILE LAST UPDATED: 6 JUL 2000 (20000706/UP). FILE COVERS 1960 TO DATE.

MEDLINE has been reloaded to reflect the annual MeSH changes made by
the National Library of Medicine for 2000. Enter HELP RLOAD for details.

OLDMEDLINE, data from 1960 through 1965 from the Cumulated Index
Medicus (CIM), has been added to MEDLINE. See HELP CONTENT for details.

Left, right, and simultaneous left and right truncation are available in the
Basic Index. See HELP SFIELDS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE
SUBSTANCE IDENTIFICATION.

=> d his

(FILE 'HOME' ENTERED AT 10:33:47 ON 10 JUL 2000)

FILE 'MEDLINE' ENTERED AT 10:33:58 ON 10 JUL 2000
L1 1 S TRELL/AB,BI
L2 0 S TUMOR NECROSIS FACTOR FAMILY RELATED PROTEIN#/AB,BI
L3 53 S TUMOR NECROSIS FACTOR FAMILY/AB,BI
L4 11 S L3 AND RELATED/AB,BI

FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 10:39:33 ON 10
JUL 2000
L5 9 S L1 OR L2
L6 8 DUP REM L5 (1 DUPLICATE REMOVED)
E BROWNING J/AU
L7 264 S E3-E5
E BROWNING JEFFREY/AU
L8 192 S E1-E9
L9 456 S L7 OR L8
L10 74 S L9 AND TUMOR NECROSIS FACTOR/AB,BI
L11 26 S L10 AND FAMILY/AB,BI
L12 16 DUP REM L11 (10 DUPLICATES REMOVED)
E CHICHPORTICHE YVES/AU
L13 1 S L10 AND TRELL/AB,BI
E CHICHEPORTICHE YVES/AU
L14 70 S E2-E3
L15 3 S L14 AND TRELL/AB,BI
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)
L17 9 S L1
L18 8 DUP REM L17 (1 DUPLICATE REMOVED)

FILE 'STNGUIDE' ENTERED AT 10:52:27 ON 10 JUL 2000

FILE 'MEDLINE' ENTERED AT 10:56:01 ON 10 JUL 2000

=>

---Logging off of STN---

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Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.30 134.29

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
SINCE FILE TOTAL
ENTRY SESSION
CA SUBSCRIBER PRICE 0.00 -5.01

STN INTERNATIONAL LOGOFF AT 10:56:18 ON 10 JUL 2000